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# DISPERSAL GRADIENTS AND DEPOSITION EFFICIENCY OF VENTURIA INAEQUALIS ASCOSPORES AND THEIR RELATIONSHIP TO LESION DENSITIES (APPLE SCAB DISEASE, SPORE DISPERSAL, TRAPPING)

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# DISPERSAL GRADIENTS AND DEPOSITION EFFICIENCY OF *VENTURIA INAEQUALIS* ASCOSPORES AND THEIR RELATIONSHIP TO LESION DENSITIES

BY

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## Reading Myself

Like thousands, I took just pride and more than just,
struck matches that brought my blood to a boil;
I memorized the tricks to set the river on fire-
somehow never wrote something to go back to.
Can I suppose I am finished with wax flowers
and have earned my grass on the minor slopes of Parnassus....
No honeycomb is built without a bee
adding circle to circle, cell to cell
the wax and honey of a mausoleum-
this round dome proves its maker is alive;
the corpse of the insect lives embalmed in honey,
prays that its perishable work live long
enough for the sweet-tooth bear to desecrate-
this open book...my open coffin.

Robert Lowell

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## ABSTRACT

DISPERSAL GRADIENTS AND DEPOSITION EFFICIENCY OF *VENTURIA INAEQUALIS* ASCOSPORES AND THEIR RELATIONSHIP TO LESION DENSITIES

by

Jonathan David Kaplan
University of New Hampshire, May, 1986

The airborne dispersal of *Venturia inaequalis* ascospores and *Lycopodium* sp spores from a point source was examined in an apple orchard. Twenty-five volumetric spore traps were constructed, and a system developed that governed intervals of trap operation by leaf wetness. The estimated inoculum density was equal to several unsprayed trees, much greater than inoculum densities in well-managed commercial orchards.

Dispersal gradients were analyzed using the equation $Y = a + bX_1 + cX_1^2 + dX_1^3 + eX_2$, where Y is the log of spores trapped, $X_1$ is the log of distance from the source, and $X_2$ is a constant to relate trap location to wind direction. Statistically significant ($p = 0.05$) regressions were calculated when data for all ascospore release periods were combined, but not for individual releases. Ascospores were randomly dispersed beyond 30 m from the source, and it was proposed that beyond 30 m an equivalent inoculum source within or outside of a commercial orchard would not contribute significantly to the inoculum level in that orchard. *Lycopodium* spore gradients were steeper than ascospore gradients, possibly reflecting differences in terminal velocity.

The ratio of airborne ascospores per developing lesion on unsprayed foliage was calculated by dividing area dose (ascospores $cm^{-2}$ air) by lesion density (lesions $cm^{-2}$ leaf tissue), where area

dose = spores $m^{-3}$ x wind speed (m $s^{-1}$) x sampling period (s)/10,000. The ratio was greatest for leaves infected at the beginning of the primary season. The percentage of scabbed fruit on trees unprotected with fungicide prior to pink bud phase was not statistically greater than on trees protected for the entire primary scab season. On potted McIntosh seedlings placed in the orchard, the relationship was described by the equation $Y = 1.79 + 0.0073X$, where Y is the lesion density and X is the airborne ascospore density when the airborne ascospore density was greater than 5000 ascospores $m^{-3}$ air. Below that figure, there was no relationship.

The deposition efficiency (percentage of area dose) of ascospores deposited on coverslips affixed to leaves in a glasshouse study was 3.8, 2.5, and 0.8% at wind speeds of 0.23, 0.45, and 0.89 m $s^{-1}$, respectively. The low number of lesions recorded in the orchard was thus partially explainable by the small proportion of ascospores deposited on foliage.

# INTRODUCTION

The association of plant disease with the wind has been well known for several thousand years. Most students of any introductory plant pathology course are enlightened to biblical references to "blight and searing wind" (Deuteronomy 28:22) or Pharaoh's dream of "seven ears of grain, shriveled and thin and blasted by the wind" (Genesis 41:6). In more recent times, wind has been implicated in the annual spread of black stem rust of wheat northward from Mexico, through the United States to Canada, (101, e.g), and a recent epidemic of blue mold in Connecticut was associated with movement of the pathogen, Peronospora tabacina, from Jamaica and Cuba to Connecticut (8). The long-range transport of pathogen propagules is a fascinating aspect of plant pathology, and one that may be important in the introduction of pathogens to new areas, but for most plant diseases, the pathogen is usually endemic and present within a region.

The continuous presence and survival of many plant pathogenic fungi is dependent upon the sucessful reunion of the pathogen with its susceptible host. In agroecosystems, this reunion is usually an annual occurrence because of the large numbers of propagules produced by most plant pathogenic fungi and the high density of genetically similar host plants in a restricted area. Despite the large number of both components in these systems, a medium is still required to bring them together. For many fungal pathogens of above-ground plant parts, the medium is the atmosphere and, more specifically, the wind.

Given that the three components for infection are present, i.e.

infectious propagules, susceptible host tissue, and favorable environmental conditions, three processes are necessary to complete a wind-mediated, host-pathogen encounter. The pathogen propagule must first be released or discharged into the atmosphere by any means which propels the propagule beyond the laminar air layer bounding the surface containing the pathogen. The propagule must then be transported via the wind to the host plant, a process known as dispersal or dissemination. Finally, the propagule must penetrate the laminar boundary layer for deposition on the host plant surface. Deposition can occur by sedimentation in calm conditions or, more often, by impaction.

One pathogen which relies upon wind to renew contact with the host plant each year is Venturia inaequalis (Cke.) Wint., the causal agent of apple scab disease. In New Hampshire, the pathogen overwinters on the orchard floor as undeveloped pseudothecia in leaves infected the previous growing season. The pseudothecia mature during early spring, and matured ascospores are discharged predominantly during daytime rains (78). Ascospores, the primary inoculum for this disease, are then carried by the wind to expanding, susceptible leaf tissue where infection occurs.

Infections caused by ascospores are referred to as primary infections, and the entire period throughout the spring when primary infections can occur is the primary season. Although its exact duration is influenced by weather conditions, the primary season in New Hampshire usually begins in mid-April and ends the first or second week in June. Conidia produced in lesions resulting from primary infections comprise the secondary inoculum, which is splash-dispersed

within the canopy. Conidia can initiate secondary infections on fruits and leaves throughout the remainder of the growing season and, under suitable conditions, can lead to a substantial build-up in scab incidence. Because of the potential for several cycles of secondary infections, apple scab disease is considered a compound interest disease (128) because of the rapid rate at which disease incidence can increase given the proper conditions.

Control of apple scab disease has been accomplished predominantly with fungicides, especially in New Hampshire where more than 6000 ha are planted to McIntosh and Cortland (4), ccultivars which are highly susceptible to V. inaequalis. The strategy for most spray programs is to prevent primary infections from occurring, thereby minimizing the risk of secondary infections. The rising cost of pesticides combined with increased environmental awareness have mandated more efficient and judicious use of these chemicals. The development of quantitative models and values to describe all components of the apple scab disease cycle is essential for the establishment of new disease management strategies. How quantification of these components can aid in management of primary scab has been recently discussed by MacHardy and Jeger (80).

To date, work has been completed that describes quantitatively the development of V. inaequalis from its overwintering state through spore discharge (85), and models have been developed that predict levels of ascospore maturity within a population (35,58) and the length of the primary season (79). Instrumentation has been developed to aid growers to monitor environmental conditions and determine whether or not infection periods have occurred (62,81). In contrast,

there has been very little work to quantify either the dispersal and deposition of ascospores of V. inaequalis, or subsequent infection and lesion development. These aspects of the disease cycle are essential if the pathogen is to unite with the host for another growing season.

The research presented in this dissertation was conducted to quantitatively describe the above phases of the disease cycle. Chapter II reviews literature pertinent to research methods and components of the apple scab disease cycle studied. Research described in Chapter III was conducted to examine the dispersal of ascospores of V. inaequalis in an orchard environment. The studies described in Chapter IV were designed to relate airborne ascospore dose in an orchard to lesion density resulting from those spores. The final chapter consists of glasshouse experiments to relate airborne ascospore densities to the density of ascospores deposited on apple leaf surfaces. Throughout the field studies, attempts were made to maintain realistic situations which are most similar to those encountered under agricultural growing conditions, with full knowledge that adherence to reality can lead to concessions in the development of quantitative relationships. At the same time, the author believes that those results which are obtained under such guidelines are easily adaptable to an agricultural system, such as an orchard, to aid the grower in making management decisions.

# CHAPTER II

## SPORES IN THE LITERATURE

This review of the literature has four sections. The first section describes the different types of traps which have been designed to sample the air spora. The second section reviews studies that have utilized spore traps. The third section presents research concerned with the dispersal and deposition of spores. The fourth section reviews spore dispersal studies involving Venturia inaequalis.

# I. THE TRAPS

A number of devices and techniques have been developed to sample the air spora. They rely on the properties of either impaction or sedimentation to trap spores, and the method of trapping varies from simple to complex depending upon the needs of a particular study.

Among the most simple devices is a gravity slide technique (46), in which a microscope slide is placed horizontally along a surface and spores land on the slide via sedimentation. The slide can be coated with some sticky substance, such as silicon or vaseline, to enhance spore retention. The slide is then examined microscopically to estimate the number of spores deposited on a specific area. Two main advantages of the gravity slide technique are that it is inexpensive and very simple to use in any situation. Two disadvantages are the small area sampled and the trapping efficiency, although efficiency is greatly improved as wind speeds increase (46).

Sticky glass slides can also be used in a vertical position to trap airborne spores via impaction. In this way, slides have been used to monitor the appearance of wheat rust uredospores (102) and to study the dispersal of Lycopodium spores (110,131). Whereas horizontal slides give an indication of spore deposition density, vertical slides monitor the airborne spore density. Airborne spore density can also be studied using sticky glass rods or cylinders. Varying the size of a rod or cylinder alters its trapping efficiency (43).

Another simple trapping surface is the petri plate which, like a glass slide, can be used to study sedimentation or impaction. Plates may contain a selective medium to monitor the presence of specific organisms that will grow on the medium. This method is used for quantitative studies because the number of colonies that develop reflects the number of spores deposited, and the density of deposited spores is proportional to the density of spores in the air. This technique was used to study dispersal of Fusarium subglutinans (Wollenw. et Reinking) Nelson et al., the causal agent of pine pitch canker (11). When protected petri dishes were placed in heavily infected plantations, in natural stands low in disease, and in clear cut areas, an average of 17.9, 3.1 and 2.0 colonies per plate were recovered, respectively. Petri plates and glass slides, because of their simplicity, have also been used to sample the air spora in unusual places such as the atmosphere 1500 meters above ground level (46,92,111).

An important concept to be considered for any spore trap is the trapping efficiency of the device. Efficiency can be defined by the equation E = Trap dose/Area dose, where trap dose is the actual number of spores deposited and area dose is the number of spores flowing through an imaginary unit area, such as a 1 cm square at right angles to the trapping surface (assuming the surface is horizontal, such as a leaf)(45). The trapping efficiencies of surfaces will vary with the size, shape, and topographical features (e.g. pubescence, contours) of the surface, orientation in relation to the direction of the wind, and the speed of the wind (46).

The trapping efficiencies of glass slides, rods, cylinders and

petri plates that rely on gravity or wind for deposition and impaction are low, especially at low wind speeds. In fact, these traps theoretically have a trapping efficiency of zero under calm conditions (46). The trapping efficiency must be increased for more concise and quantitative studies of air spora. Volumetric spore traps that sample a known quantity of air by suction have an increased trapping efficiency. Two primary advantages of suction traps are 1) they increase efficiency of impaction by accelerating a narrow air stream onto a surface, and 2) some can indicate the hour in which the spores were trapped, thereby enabling correlation between environmental factors and the presence or absence of spores in the atmosphere (52).

One of the first volumetric spore traps was designed by Frey and Keitt (33) to study the epidemiology of apple scab disease. They had originally tried to devise a trap utilizing a static flow tube. Air was drawn through a glass tube and the electrostatic properties of the spores were utilized to remove them from the air via electricity and deposit them on the glass. Unfortunately, the spores tended to stick to the sides of the tube. They eventually devised an apparatus in which a vacuum pump drew air through a transparent membrane at 20 liters $min^{-1}$. The spores were deposited on the membrane and then counted with a microscope. In this way, they monitored the presence of Venturia inaequalis ascospores in the air.

Another volumetric sampler is the Manning slit sampler (12) designed to trap bacteria. A measured amount of air is drawn through a fine slit and then impinged upon a slowly revolving plate of culture medium. The plate is then removed and incubated. By using a selective medium, the sampler can detect a specific fungus or

bacterium. This approach was taken by Dimenna (24) in a medically orientated survey of the air spora around a hospital.

A similar sampler is the Anderson sampler (6) which, through the use of various filters, collects and separates spores and bacteria of different sizes. The sampler has six levels, each containing a petri plate with sterilized medium. Air is drawn through the apparatus and spores are deposited on the plate, with the largest spores trapped on the upper plate and spores of decreasing size deposited at each successive level. Selective media have also been utilized in the Anderson sampler to enhance the growth of specific fungi (95, e.g.).

An entirely different trap utilizes centrifugal force to impact spores on a surface. An example is the cyclone collector designed by Ogawa (93). The cyclone trap can draw up to several hundred liters of air per minute. Spores are deposited in a flask or vial, offering the advantage of collecting viable spores and obtaining cultures from them. However, at high intake speeds there is some mortality (125) which must be considered when conducting quantitative studies.

The first recording volumetric trap was designed by Hirst in 1952 (52). Fromme (34) had designed a recording trap in 1918, but it was not volumetric. The Hirst trap was designed to sample isokinetically, with the rate of intake equal to the speed of the wind. Wind tunnel tests using spores of Lycopodium clavatum (L) revealed trapping efficiencies between 93.8% and 62.4% as the wind varied from 1.5 to 9.3 meters $s^{-1}$. The trap was built with a wind vane so that it always faced into the wind. Spores are deposited in bands on a moving slide, with each band representing one hour's deposit. In this way, one can determine the time of day when specific spores are being deposited, a

distinct advantage for many studies. The Hirst trap (C.F. Casella & Co, London, N.1 England) and the similar Burkhard trap (Burkhard Manufacturing Co., Rickmansworth, Herts, England), which traps spores on a drum that revolves once every seven days, are commonly used to monitor spores and other airborne particles.

The Kramer-Collins spore sampler (69) is similar to the Hirst trap. This trap was designed to sample intermittently or continuously, with spores being deposited on a slide moved by a spring-wound clock. The trap is useful for studying the periodicity of spore release, and is isokinetic when drawing in air at 22.7 l $min^{-1}$ with winds of 0.0 m $s^{-1}$. Recently a new, similar trap was designed that has a 7-day revolving drum upon which spores impinge (68). Wood and Schmidt (132) built a trap similar to the Kramer-Collins sampler to study release of bracket fungal spores. The spores were caught on a strip of 35mm film, of which various portions were exposed by a sliding orifice. This trap, however, relied on sedimentation to sample spore release.

Another trap design utilizes inertial impaction to trap spores. The first and most popular model is the rotorod sampler (Ted Brown Assoc., Los Altos, CA), originally designed by Perkins (96). The trapping surfaces are two sticky arms set on brass rods. These rods spin at approximately 2400 RPM to sample air at a rate of 120 liters per minute (46). Advantages of the rotorod sampler include low cost, light weight, large sampling volume, and battery operation for sampling in remote areas. A disadvantage arises when used for long periods of time, as the rotorods tend to collect too many particles, making the task of counting specific spores or pollen much more

difficult.

Concerning rotorods, Edwards (26) emphasized the importance of collection efficiency which must be considered when utilizing rotating arm impaction samplers. The efficiency varies with the size of the particle, and may cause erroneous estimates of spore concentration. He developed an equation for computation of a particle parameter, p, which is unique for each size of particle. If p is greater than 10, than one can assume an efficiency of 100%. A chart devised by Noll (26) should be consulted to determine the actual efficiency for particles with p values less than 10. Appropriate modifications of the collection surface size should then be made to increase efficiency.

The rotoslide (94) is a trap similar to the rotorod, except particles are impacted on microscope slide edges coated with silicon grease which rotate at approximately 1500 RPM. The trap was designed by Ogden and Raynor to study particle dispersal into and within a forest.

As mentioned, an important characteristic of any spore trap is its efficiency of collection. The degree of efficiency desired will depend on the nature of the study. A less efficient trap such as a slide might be used when the particle concentration is high, especially if qualitative rather than quantitative results are the primary objective of the study. More efficient traps must be used when the concentration of particles is low. For example, Hirst and Stedman (53) compared the detection thresholds of glass slides inclined at a $45^{o}$ angle, to that of a Hirst trap. Thresholds for spores of Lycopodium and Ustilago sp were, respectively, 260 and

13,000 spores $m^{-3}$ of air for the slides, and 2 and 2 spores $m^{-3}$ for the Hirst trap, at a wind speed of 1.1 m $s^{-1}$. The efficiency of the sticky slides increased dramatically as the speed of the wind increased, but never approached that of the volumetric trap.

Several other studies have compared the relative efficiencies of various traps. Leuschner and Boehm (74,75) devised an individual pollen collector to be worn by allergy patients. The collector consisted of a vaseline-coated slide which was placed in a white, plexiglass device designed to simulate the shape of a human nostril and eye socket. They compared the individual pollen collector to a Burkhard trap and found no qualitative differences between them, although there were quantitative differences in the ratios of certain types of pollen. However, the pollen collector was useful because it could detect "clouds" of pollen, thus providing a qualitative pollen map of the patient's travels.

Eversmeyer et al. (29) compared the trapping efficiency of their 7-day drum sampler to the original Kramer-Collins sampler and the Hirst trap. They released 50 mg of Puccinia graminis Pers. uredospores in a wind tunnel and simultaneously operated the three traps. Glass rods and rotorods were included to monitor the uniformity of the spore cloud. The three traps were similar in trapping efficiency, and the trap one would choose would depend upon the nature of the study. The Kramer-Collins trap is more versatile and readily adaptable to study the air flora in a variety of habitats and locations. The Hirst trap is suited for use at ground level, and is limited by its bulky size (29).

Tate et al. (125) conducted field and wind tunnel studies to

compare the efficiency of their cyclone trap to the Hirst trap (the standard for the field) and a Cascade Impacter. The cyclone trap was more efficient than either of the other two traps, especially at low spore concentrations, and in one field test caught six times as many spores as the Hirst trap. A beneficial feature of the cyclone spore trap is its ability to collect viable spores in the field, although spores of Monilinia fructicola (Wint.) tended to stick to the walls of the collector.

Sutton and Jones (118) compared rotorods, a Burkhard trap, glass rods, and slides to determine the relative abilities of these traps to trap ascospores of V. inaequalis. Their purpose was to find the trap best suited for monitoring ascospore discharge in a warning system for growers. They recorded the total catch by each trap as a percentage of the season's total. The glass rods were 1.5 to 4 times more efficient than the glass slides, but neither trap was comparable to the Burkhard trap. The rotorods were as efficient at trapping ascospores as the Burkhard trap when placed 0.45 m above the ground at all but low levels of ascospore concentration. Inconsistencies at the low levels were possibly due to the fact that there were more rotorod traps used in the study. The rotorods were better suited for routine monitoring of V. inaequalis ascospores because of their low cost and ease of use, but it was suggested that they be used in orchards known to contain high levels of inoculum.

Economics was an important factor in the development of two other spore traps used to monitor V. inaequalis ascospores. Gadoury and MacHardy (36) designed a trap similar in design and function to the Burkhard trap, at 10% of the cost. The major cost saver was the use

of polyvinyl chloride (PVC) instead of stainless steel and cast alloy. The trapping efficiency of the PVC trap was tested by trapping conidia of M. fructicola in laboratory studies and by trapping ascospores of V. inaequalis in orchard studies. In both situations, the trapping efficiency of the PVC was equal to that of the Burkhard trap. The traps have been used successfully in epidemiological studies at the University of New Hampshire.

PVC is also the predominant material used in the trap designed by Zuck (135,136). This trap cost $50 to construct in 1984, and has been used by growers in the state of Maine in their management programs for apple scab. Twenty-five similar traps were constructed and utilized in this study (Appendix A).

If cost is not a consideration, one might consider the trap designed by Gottwald and Tedders (41). Although this trap cost approximately $75.00 to build, it was developed to be placed on a miniature drone (remotely piloted vehicle) that would cost at least $700 - $1000 to build, an investment lost with the first confrontation with wind shear and other crash-inducing phenomena. It would seem that the scale of agriculture in New England prompts Yankee ingenuity to design low cost spore traps, while researchers in regions with large scale agriculture have sophisticated, electronic model airplanes to house their spore traps!

In conclusion, there is a large variety of spore traps available for sampling the air spora. The choice of a trap will depend upon the individual needs of the researcher as well as any financial constraints which might be imposed. The choice of trap in this study was based upon the ease with which it could be built, the low price,

and the ability to determine quantitatively the density of airborne ascospores.

## II. SELECTED STUDIES THAT UTILIZED SPORE TRAPS

Webster's New Collegiate Dictionary (133) defines dispersal as "...the process or result of the spreading of organisms from one place to another", and disseminate as the process of "...dispersing throughout" or "...spreading abroad as though sowing seed". In preparing this review of studies involving spore trapping, many articles have been retrieved because the title included the word dispersal or dissemination (17,30,32,56,59,95,103,116,119,129, e.g.). Upon reading the articles, it was discovered that what was being reported were, more often than not, patterns and periodicity of release; i.e. when the spores were being dispersed, not where they were dispersed. Some of these studies are discussed briefly as a prelude to a discussion on studies conducted to model spore dispersal.

The Hirst volumetric spore trap has often been used to study spore release and dispersal of many important plant pathogens. Sreeramulu (109) examined seasonal and diurnal periodicities of several fungi. On a seasonal basis, he found that pathogens were more prevalent early in the season when crops were actively growing, and saprophytes were more common later in the season as crops matured. He noted diurnal peaks for organisms representing several genera of fungi, including Ustilago, Erysiphe, Helminthosporium, Botrytis, Polythrincium and Epicoccum.

Gregory and Stedman (48) studied airborne "dispersal" of Ophiobolus graminis Sacc., the pathogen causing take-all disease of wheat, by placing a Hirst trap over wheat stubble prior to plowing. Take-all is a root disease, thus the aerial dissemination of the pathogen is generally considered unimportant. However, ascospores of O. graminis were trapped, with a peak of 3700 spores $m^{-3}$ air. The presence of ascospores was always associated with rain, as spores were never caught on rainless days. A minimum of 2.5 mm of rain was necessary to induce release, and with prolonged rains the supply of mature spores was eventually exhausted. The pattern of release by O. graminis was similar to that of Venturia inaequalis (13), although liberation following commencement of precipitation occurred more quickly (1 hour vs 3) for O. graminis. Interestingly, ascospores of O. graminis have not been shown to initiate infections.

Sclerotinia fructicola (Wint.) Riehm. has a diurnal periodicity of ascospore release similar to that of O. graminis and V. inaequalis, but differs from these fungi in that the ascospores are a component of the dry air spora in orchards. However, a nighttime dew might be required for normal function of the apothecia (124).

Meridith (88) examined release of conidia by Deightoniella torulosa (Syd.) Ellis, the causal agent of fruit spot of bananas. A single Hirst trap placed centrally in a plantation demonstrated that spore release was on a diurnal cycle, with release occurring between 0600 and 0800 hours coincidental with a drop in relative humidity. This periodicity correlation with relative humidity is similar to another moniliaceous fungus, Drechslera turcica (Pass.) Subram. & Jain, the causal agent of northern corn leaf blight (72). Low

relative humidity also favored dispersal of Podosphaera leucotricha (Ell. & Ev.) Salm. in apple orchards (119).

Botrytis cinerea Pers. is a ubiquitous pathogen, to the point where it has been caught above the Atlantic Ocean (46). Conidial release in raspberry plantations followed a diurnal periodicity, with a peak concentration at midday and a smaller peak in the evening (59). Despite its common occurrence in the atmosphere, numbers of spores in areas where B. cinerea is not a problem compared to numbers where it is a problem led Jarvis (59) to suggest that locally produced inoculum is responsible for infections by this fungus.

There are several fungi whose spore release does not exhibit diurnal periodicity. One fungus is Alternaria dauci (Kuhn) Groves and Skolko, the pathogen causing leaf blight of carrots. Conidia were trapped more often during daytime hours, but the greatest correlation was with leaf wetness (116). Conidia of Diplodia gossypina Cke., one of several fungi associated with boll rot of cotton, were usually trapped at night or early morning, when the relative humidity was near 100% (103). Perhaps more significant is that spore levels of this and other associated fungi are closely related to the shedding of flowers, bolls and squares from cotton plants.

A fungus requiring rain for spore release is Guignardia bidwellii (Ell.) Viala & Ravaz, the causal agent of black rot of grape. The maximum release of ascospores coincided with the maximum susceptibility of the vines (32). A phenological relationship between host and pathogen also seems to occur with Venturia inaequalis and apple trees, as the peak maturation of spores usually coincides with the period of bloom, when a maximum of susceptible tissue is present

(16,120).

Not all fungi have release patterns as simple as those just given. Sutton and Jones (119) studied the effects of individual environmental factors on release of powdery mildew conidia in Michigan apple orchards. They distinguished three types of spore dispersal patterns. Type 1 was a diurnal pattern, with densities increasing in the morning after leaves were dried and remaining high until 2000 to 2200 hr. Fifty-five percent of the spores trapped fit this catagory. Thirty-five percent of the spores trapped were in type 2, i.e., spores were abundant throughout the day with no apparent periodicity. Type 3 dispersal consisted of a low percentage of spores caught during rainfall, when airborne spore density would be very high initially and then fall to very low levels. The authors concluded that individual environmental factors may be important, but the interaction of these factors has the greatest effect on dispersal.

Faulkner and Colhoun (30,31) investigated the airborne dispersal of conidia of Leptosphaeria nodorum Muller. The fungus attacks gramineous crops and was not considered likely to be dispersed through the air because its spores are produced in pycnidia and exuded in a cirrus. A spore trap was developed based on the pre-impinger trap developed by May and Druett (86). The trap collected pycnidiospores of the fungus in an aqueous solution of 12.5% glycerol. The spores were concentrated by centrifugation, resuspended, counted on a haemocytometer to determine airborne concentration, and then seeded onto Czapek-Dox V-8 agar to determine their viability. Viable conidia were trapped 2 m above a growing crop, 2 m above stubble of a crop, and 1 m downwind from a crop, demonstrating the potential for long

distance aerial dispersal of the pathogen. The authors' conjecture that sphaeropsidacious fungi might be aerially dispersed was supported by Carnegie (17). He trapped viable conidia of a similar fungus, Phoma exigua Desm. var foveata (Foister), the causal agent of potato gangrene disease, 100 m downwind from the nearest source of spores, a fallow potato field.

Tisserat and Kuntz (126) placed rotorods 6.2 m above the ground to trap the conidia of a third sphaeropsidacious fungus, Sirococcus clavigignenti-juglandacearum Nair, et al., a fungus which is a speaker's nightmare as well as the causal agent of Butternut canker disease. Finally, Bertrand and English (10) designed their own funnel traps and detected conidia of Valsa leucostoma (Pers:Fr) Fr. 78 m from an inoculum source.

In conclusion, spore traps have been used in a number of ways to study the ecology of plant pathogens and epidemiology of plant diseases. Their use has enabled pathologists to "see" pathogen propagules as they are disseminated towards the host plant. Improved vision of dissemination processes has enabled pathologists to recommend control measures for some diseases more effectively and efficiently.

# III. MODELING OF SPORE DISPERSAL

Modeling of spore dispersal requires the integration of physical theories of particle dispersion with recorded events in the field via complex mathematics. Ideally, a spore dispersal model should predict the exact number of spores found at any distance from a known amount of spores released at the source. All that would be required would be the wind direction, speed, surface topography, and meteorological parameters such as rain, temperature, and relative humidity. As with other models of environmental phenomena, the theory and reality of spore dispersal are difficult to unite!

Disease gradients have been used as indicators of dispersal gradients. Newhall (92) studied the disease gradient of onion downy mildew, caused by Peronospora destructor (Berk.) Casp, from an abandoned garden that was the inoculum source throughout a valley. A decrease in disease incidence with distance downwind from the inoculum source demonstrated that conidia were airborne. Viable conidia were also trapped on water agar from an airplane. Wilson and Baker (130) used disease gradients to correlate the spread of brown rot blossom disease of peach, caused by Sclerotinia laxa (Alderh. & Ruhl) Honey, with environmental factors influencing the dissemination of spores in an orchard. Unsprayed trees in the middle of the orchard provided a source of inoculum. Because of weather conditions, all blossom

infections occurred during one infection period in both 1939 and 1940, facilitating the correlation of disease spread with distance from the inoculum source.

In both years blossom damage decreased with distance from the inoculum source. As the distance increased to 22, 44, 66, and 88 feet from the source, per cent infection dropped to 39, 21, 12, and 6.5% of disease at the source in 1939, and 55, 40, 28, and 23%, respectively, in 1940. The difference between the two years was directly related to an increase in wind speed in 1940. Twenty of 32 upwind trees had blossom infection (less than 1%), indicative of background inoculum or upwind dispersal.

Dispersal gradients were used to establish the etiological relationship between Podosphaera leucotricha (Ell. & Ev.) Salm., the causal agent of powdery mildew of apple and possibly rusty spot of peach (100). It was alleged (22,84), but not confirmed, that the same fungus causes both diseases. Disease incidence along a transect or straight line varied such that plotting the number of infections (y) against distance traveled (x) produced a straight line, with the slope of the line (b) the measured disease gradient. The linear regression of $\log_{10}y$ and $\log_{10}x$ was calculated using the equation $y = a + bx$, where $y = \log_{10}$ of the percent of infected fruit at x, the the $\log_{10}$ of the distance from the source. The inoculum source was an apple orchard with approximately 1% infection, and disease was measured 8, 24, 40, 56, 72, and 90 meters downwind into an adjacent peach orchard. Regression analysis provided significant correlation ($p = 0.01$) between the measured disease gradient within the peach orchard and distance from the source apple trees, with a greater correlation when

disease was recorded as the number of lesions per fruit. Their data, when combined with the knowledge that rusty spot disease is only found on peach trees grown near apple trees with powdery mildew, provided strong evidence that P. leucotricha was indeed the causal agent of rusty spot disease of peach.

Lin (76) analyzed disease gradients of sorghum downy mildew, caused by Peronospora manshurica (Naum.) Syd. ex Gaum. Inoculated plants were placed in the extreme upwind corner of a field, and the amount of disease was recorded weekly at various points along several arcs which were 3.0, 6.1, 12.2, 18.3, and 27.3 m from the initial infection focus. A linear regression of disease incidence against distance was calculated using the same equation used by Ries and Royse (100). The value of b (representative of the steepness of the disease gradient) was -0.41, -0.32, -0.08, and 0.03 after 30, 44, 58, and 72 days. The flattening of the gradient was coincidental with uniform increase of disease incidence throughout the plot. Lin had also placed several rotorod samplers above the crop at 3.0, 12.2 and 27.4m from the inoculum source, but he made no attempt to correlate the numbers of spores trapped with disease severity or meterological records.

Disease gradients for peach powdery mildew, caused by Oidium sp., were also analyzed to determine the distance required to isolate peach trees from wild roses, the initial source of inoculum (63). Disease was measured at several distances into an orchard that had a strip of roses adjacent to it. The data were used to compare the models of Gregory (44) and Kityosawa and Shiyami (67) (see below). The Gregory model had the best fit, although it over-predicted the amount of

disease near the source of inoculum. The authors concluded that 27 to 71 meters were required for effective isolation of peach trees.

The models used to analyze disease gradients in the above studies are two of several models developed to estimate the spatial distribution of inoculum or infection around a source. These models were all recently examined by Lambert (70) et al. who developed a general model for disease gradient analysis. A major point of Lambert et al. (70) was that although the slope of each model was variable, allowing for different gradients, the shapes of the curves themselves were all fixed, preventing adequate analysis of data sets with intermediate or variable shapes. Based on the models of Gregory ($y = a + bx$) (44), Kampmeijer and Zadoks ($y = a/2\pi s^2 \exp(-x^2/2s^2)$) (70), and Kiyosawa and Shiyomi ($y = a\exp(-bx)$) (67) Lambert, et al. (70) developed the general model, $y = \exp(-bx^n)$, for use in disease or dispersal gradient analysis, where y = lesions per plant, b is the slope of the gradient and x is the distance from the source.

The general model was applied to disease indexing data gathered after placing a single rice plant infected with Pyricularia oryzae Cav. in the center of a 2.4 m x 8 m plot of rice. Five models were obtained that varied in the degree of correction for wind direction. Correcting for wind direction significantly improved the accuracy of the general model, with a maximum $R^2$ value of 75.7. Best fit was obtained when n = 0.284. They advocated use of the general, flexible model because it was more accurate at distances closer to the source. The previous models tended to be more accurate at intermediate distances and less accurate nearer the source, where most infections occur.

A visual alternative to measuring spore dispersal is utilizing a marker such as smoke. Zadoks et al. (134) used a smoke generator to release isothermic smoke puffs above a barley crop. Sequential pictures taken of the puffs as they spread through the field revealed two types of air movement. One type acted to extend the smoke clouds and the other displaced the entire cloud as a whole. Eight of ten puffs moved upward and two moved toward the ground. Interestingly, half of the puffs moved against the wind, which was very light at the time.

Wilson and Baker (131) generated smoke puffs in a peach orchard by bubbling HCl through a solution of NaOH, and reported that the greatest dispersal was in the horizontal plane. They developed a model utilizing a cone as a basic geometric model, with the apex of the cone being the particle source (130,131). Dispersion would occur horizontally down and across the wind and vertically, with the degree of dispersion being influenced by the velocity of the wind. At low wind speeds, the degree of dispersion decreases rapidly and the cone is more contracted. At high wind velocities, the cone is elongated, with equivalent spore densities occurring at a much greater distance from the source than at lower wind speeds.

To verify the model a wooden frame 15 x 7 feet was constructed with the center of the widest part 7.5 feet above the ground. Oil-coated slides were placed on the framework so that there were five slides at specified distances from the source (apex of the cone), with one slide on each of four outer boundaries and the fifth in the middle of the cone. Spores of Lycopodium sp were released in puffs from the apex. Vertical distribution was studied by placing slides at 12 inch

intervals above and below the center of the framework.

The standard deviations of the distribution of spores were used as a measure of dispersion. Two models, $sd_{vx} = ax^p$, and $sd_{hx} = bx^q$ were developed to describe vertical and horizontal distribution, respectively, where a and b are constants dependent on wind speed, p and q are numbers between 1/2 and 1, x is the distance from the source, and $sd_{vx}$ and $sd_{hx}$ are the standard deviations of dispersion in the vertical (v) and horizontal (h) plane, respectively. The experimental data approximated the geometric model, though the cone was expanded with greater densities toward the middle and horizontal, especially nearer the source. The experiment was repeated in a field and an orchard, with the spore source on one side of a tree and the trapping surface on the other side. The spores were more widely dispersed (as measured by standard deviation) in the orchard than in the field.

One of the more important spore dispersal studies was completed by Gregory et al. (47), utilizing spores of Lycopodium clavatum. Spores were released from a point source in a flat field and trapped with numerous miniature suction traps placed at specified distances up to 10 meters from the source. Gregory (42) had previously developed a model of spore dispersal based on Sutton's theory of eddy diffusion (117), and this study was designed to test that model. The increase in horizontal cross-wind standard deviation with distance from the source was computed, as had been done by Wilson and Baker (131), and was found to fit Sutton's theory. The experiment was also repeated with similar results using spores of Ganoderma sp, and it was concluded that the size of the spore had little effect on the pattern

or gradient of dispersal, at least within the short distances used.

Sreeramulu and Ramalingam (110) conducted a study associated with Gregory et al. (47). Spores of Lycopodium sp were trapped on sticky slides placed at 5 meter intervals up to 30m from the source, thus sampling a larger area than Gregory et al. (47). Conditions were much more turbulent in this study than in Gregory's et al. (47), but data fit Sutton's theory of eddy diffusion and the mathematical model developed by Gregory (42) based on Sutton's theory.

The theory and equations developed by Gregory (42) are complicated. One equation defines the density of spores at any distance from a point source as follows:

$$X = \frac{Q}{(\pi)^{3/2} C^3 (ut)^{3/2} m} \cdot \exp \frac{r^2}{C^2 (ut)^m}$$

where Q equals the quantity of particles liberated; C equals the coefficient of diffusion; X is the distance travelled; m is a parameter greater than 1 and less than 2, based on turbulence; r is the distance from the center of the spore cloud; u is the mean wind velocity; t is time; and y and z are parameters of vertical and perpendicular diffusion. Because m is never greater than 2, the drop in density along any axis of a point source cloud can never be more rapid than the inverse square, regardless of the amount of turbulence (42). C, the coefficient of diffusion, will vary with turbulence. Typical values of C are $0.1(m)^{1/2}$, $1.1(m)^{1/2}$, and $0.4(m)^{1/2}$ for normal, high and low levels of turbulence (117). The same variables can also be used to compute the standard deviation of the spores from their mean position, using the equation $sd = 1/2C^2(ut^{2-n})$ (117).

Another important series of quantitative studies was completed by

Bainbridge and Stedman (9) who examined dispersal of spores of Lycopodium clavatum and Erysiphe graminis DC. within and above a barley field. They used 30 miniature suction traps designed by Gregory (47). The traps were placed at several heights and at various sites downwind, up to seven meters from the spore source. A strip of Zephyr barley infected with E. graminis was the source of inoculum. Spore traps were also placed upwind of the inoculum source to determine the background level of spores. Vertical slides and glass rods were placed near the suction traps, both within and above the crop.

The greatest concentration of Lycopodium spores was near the source, but there was also rapid, upward diffusion. The area dose was always greatest at the top of the crop, but 7 m from the source the spore density was uniform throughout the crop. With E. graminis the concentration of conidia detected beyond 4 m and above 0.5 m did not differ from the background spore density. Stronger winds resulted in greater spore densities at greater heights. The rapid decline in spore density was related to the high density of the crop, which intercepted spores and also kept them within the canopy, preventing redistribution.

Data from Bainbridge and Stedman's studies (9) were used to test the validity of a two-dimensional model of movement and deposition of spores within and above a barley crop (73). This model was developed because it was felt that earlier models described by Gregory (42), based on the diffusion equations, did not apply to spores released within a crop canopy, where the majority of these spores are redeposited at short distances, with few escaping into the atmosphere.

Also, the diffusion models had been developed and tested with conditions of open air, and the vertical parameters y, z, in Gregory's model were not defined.

Three inputs are necessary to use Legg and Powell's model (73): 1) the initial spore concentration profile 2) the wind speed profile and 3) profiles of foliage area density with the stems, leaves and heads measured separately. The output includes a spore concentration profile at any distance downwind, the horizontal and vertical flux density, and the rate of spore deposition on leaves, stems, heads and the soil surface, at any distance from the source.

The model worked well to predict the dispersal of spores of Lycopodium sp, and indicated that sedimentation was the major cause of deposition in the lower half of the crop. Impaction occurred at all heights, but was most common in the upper canopy. The model did not predict the rapid decrease in the concentration of E. graminis spores with increasing distance from the source. It was postulated that the steep gradient in spore concentration was due to a greater terminal velocity of the spores than anticipated, caused by spores clumping together. Aylor (7), however, noted that spores released passively by the wind had an initial velocity that is faster than the wind speed. When the model was modified to accmomodate this phenomenon, it correctly predicted the concentrations of E. graminis conidia that had been recorded by Bainbridge and Stedman (9) and supported the calculated proportions of spores, 0.02 and 0.1 of the area dose, trapped by glass rods and slides (9).

Stedman (113) conducted studies on splash droplet dispersal in wheat crops and bean fields (112) using a fluorescent marker.

Droplets were allowed to hit a target, situated at different heights in the canopy, to generate smaller droplets. Droplets were caught on strips at ground level and on glass rods elsewhere or on the plants themselves. Also, fluorescein-treated spores of Lycopodium sp were released to compare dry air release with splash droplet dispersal of spores of Lycopodium sp stained with crystal violet.

In the wheat field, deposits from splash droplets were detected up to 16 cm from the source at a height of 100 cm above the ground. The greatest deposits occurred on the stems and upper leaves. Movement within the wheat crop was restricted by the crop itself. All the gradients in the field were significant linear regressions of the log of deposits on linear distance, with the steepest gradients occurring when the target was low, i.e. <10 cm.

In the bean field, deposition was compared for 4 types of crops: 1) a leafy crop, height 50 cm, 2) a leafy crop, height 125 cm, 3) a partly defoliated crop, height 125 cm and 4) a defoliated crop, height 125 cm. Droplets were recorded up to 2.5 m downwind of the source in a tall, dense crop, and up to 5 m in a defoliated crop. Droplets were detected over grass 16 m from the source. Variation between the fields was due mainly to the filtering action of the plants, but less wind speed in the bean field also influenced the droplet movement. Very few spores of Lycopodium sp were trapped beyond 2 meters.

Stedman (114,115) also conducted splash droplet studies with spores of Rhynchosporium secalis (Oud.) Davis, the causal agent of leaf blotch of barley, from wet and dry surfaces. Splash dispersal during rain was studied using several modified rotorods placed at 20 cm intervals from 0 to 100 cm above the ground in a 4 $m^2$ plot of

barley (114). Variation in the number of spores trapped was great, and was not related to the duration, quantity, or intensity of the rainfall. Spores were trapped in the lower canopy early in the season. As lesions developed throughout the crop, spores were trapped higher in the canopy, but there were always greater numbers at lower heights. Similar vertical gradients have been reported for Erysiphe graminis in wheat and barley crops (71), where concentrations of spores below 180 cm above the ground were always much greater and more uniform than concentrations above 180 cm. The number of lesions recorded by Stedman (114) could not be related to the estimated spore concentration.

The significance of the second study (115) was that dry spores could be liberated by a droplet of water by the "tap and puff" process. The spores would actually float on the surface of the water. Pathogenicity tests demonstrated that infection could be initiated by spores thus dispersed.

Splash dispersal by Septoria nodorum Berk. was investigated by Griffiths and Ao (49). Conidia were caught in small sampling tubes with a plastic funnel on top, filtered through an oxoid membrane, and counted. Four plants were inoculated in each of 56 2 x 1 m plots. Spores were caught up to 0.5 m from the source and at heights up to 40 cm. Significant disease incidence was noted up to 2 m, seemingly a small distance, but the pathogen could generate an epidemic with secondary cycles.

The previous studies have emphasized short-range dispersal. A few studies have quantified spore dispersal for intermediate distances up to several hundred meters. One study used gravity slides at

distances up to 100 m to study horizontal diffusion by urediniospores of the groundnut rust fungus, Puccinia arachidis Speg. (83). Spore density decreased gradually from the source (infected plants), but some spores were caught at 100 m. Ooka and Kommedahl (95) were able to isolate Fusarium moniliforme from wind-blown soil on top of snow 400 m from corn stalks that were the inoculum source.

Eversmeyer and Kramer (28) did a more quantitative study using Kramer-Collins volumetric spore samplers at distances of 60, 120, and 180 m from a source plot of wheat to monitor dispersal by Puccinia graminis f.sp. tritici and P. recondita Rob ex Desm. f.sp. tritici. There were no significant differences in spore concentrations at each distance downwind from the source plot. A simple regression equation, $\log Q_x = \log Q_o + bx$, predicted the number of spores collected, where $Q_o$ is the initial spore concentration determined by a spore trap within the source plot. The slope, b, of the line was -0.006 for P. recondita and -0.004 for P. graminis, showing great similarity for the dispersion patterns of the urediospores of both fungi.

Most models for spore dispersal have been developed and verified with experiments in open fields or above and within field crops. Only Wilson and Baker (130) have conducted studies to model spore dispersal in an orchard. Some studies have been done in forests to quantify spore dispersal (25,98,99).

Edmonds and Driver (25) examined the dispersal of Fomes annosus (Fr.) Karst. and ZnCdS fluorescent particles in a pine forest. Particles were released under dry conditions during both night and day and trapped with 25 rotorod samplers arranged in a grid and placed 1 m above the ground. Rotorods were also placed at heights of 5, 10, 15

and 20 m for some releases. Particles were released in a cleared area as well as a heavily wooded area. Data were analyzed using Chamberlain's dispersal model (19) for a continuous ground-level point source of particles. Particles released during the night and early morning had the least amount of vertical and horizontal dispersal due to the development of temperature inversions that prevented vertical mixing of the air. Particles released in the field had greater horizontal and vertical dispersal than those released in the forest. Particles were trapped up to 2 m from the source in the stable night air in quantities which were within a factor of 2 of values predicted by Chamberlain's model.

Daytime dispersal patterns were much less predictable due to increased wind speed and turbulence, both mechanical and thermal. Horizontal and vertical mixing was greater at both sites during the day, with greater dispersal in the open area than in the forest due to greater thermal mixing in the field. In the forest, particles were channeled around or between areas of dense vegetation, and entire plumes tended to move differentially into areas of low vegetation density.

Brookhaven (NY) forest was the setting for a series of experiments by Raynor et al (98,99) to study dispersal of a variety of particles. Rotoslide samplers were used to measure airborne particle densities, and greased slides were used to estimate deposition. Plumes of fern spores and ragweed pollen released in a field and blown into a forest widened greatly before reaching the forest edge, but expanded only slightly after entering the forest. Winds entering the forest had greater speeds at the trunks of trees than in the canopy.

The dispersal of particles released within the forest was influenced by the position of release and wind speed. Particles released higher up in the canopy tended to move downward and particles released close to the ground moved upward. The effect of wind was similar to that recorded by Wilson (130) with less horizontal dispersion close to the source in stronger winds. Vertical dispersion was positively correlated with wind, with greatest downwind densities measured with light winds.

The authors concluded that vertical dispersion predominated over horizontal dispersion in the forest based on the loss of particles along the centerline downwind from the source. In contrast to the findings of Edmonds and Driver (25), the vertical and horizontal components of dispersal were greater in the forest than in the field. More research is needed to clarify the discrepancies of these two studies.

# IV. SPORE DISPERSAL AND APPLE SCAB DISEASE

There have been no studies directly concerned with spore dispersal of the primary inoculum of V. inaequalis. The necessity of rain for dispersal (13,33,53) and the diurnal periodicity of the fungus (13,14,54,78,89,120) have been established, but there is no information about the dispersion of these spores. It has been suggested that they can travel long distances (1) but there is some evidence to indicate that most of the spores remain within the immediate vicinity of the orchards in which they originate (3,15,16).

Allit (3) examined the occurrence of Venturiaceous spores in Cambridge, UK from 1968 to 1972 using a Burkhard spore trap placed on the roof of a hospital. No ascospores of V. inaequalis were trapped during the four year study. Burchill (15) measured a disease gradient from a point source of scabbed leaves placed within a plot of susceptible trees, and detected no infection beyond 15 m from the source. Burchill and Hutton (16) examined the effect of phenylmercuric chloride (PMC) on suppression of ascospore production. In a commercial orchard receiving only post-harvest and pre-bud-burst applications of PMC, contamination from outside the orchard was negligible and complete scab control was achieved.

The models of spore dispersal developed in other ecosystems, as discussed here, may be inaccurate in an orchard situation because the wide spaces and numerous trees cause increased amounts of turbulence.

Only studies by Wilson (130,131) who examined spore dispersal over very short distances and disease gradients for greater distances in an orchard, and studies by Ries and Royse (100) and Kable, *et al.* (63) who examined only disease gradients from outside sources, and Corbin and Ogawa (20) who were concerned with close range droplet dispersal of *Monilinia* (*Sclerotinia*) *laxa*, have been conducted in an orchard. These studies were examined in the previous section.

A study should be undertaken to closely examine spore dispersal patterns within an orchard and to compare these patterns to those recorded in other environments. In addition, efforts should be made to relate airborne inoculum densities of *V. inaequalis* ascospores to deposition densities and lesion densities.

# CHAPTER III

## THE DISPERSAL OF ASCOSPORES OF *VENTURIA INAEQUALIS* AND SPORES OF *LYCOPODIUM* SP WITHIN AN APPLE ORCHARD

### Introduction

There has been considerable work on the maturation and release of ascospores of *Venturia inaequalis* (13,14,53,78,89,120), but virtually none on the dispersal of ascospores. Adams (1) speculated that ascospores had the potential for long distance transport, and Keitt and Palmiter (65) observed a disease gradient from an unsprayed orchard into an adjacent orchard, but, to this author's knowledge, there have been no studies to quantify ascospore dispersal. The only spore dispersal study in an orchard was completed by Wilson and Baker (130). Using glass slides, they examined dispersal of ascospores of *Sclerotinia laxa* and spores of *Lycopodium* sp in a peach orchard. Equations were fit to their data to describe airborne spore gradients and infection gradients. There have been quantitative spore dispersal studies done in wheat (28,113), barley (9) and bean fields (112), and above grass (47,110), and models have been developed to quantify observed spore dispersal gradients (7,42,73).

In this study, the dispersal of *V. inaequalis* ascospores and

spores of Lycopodium sp were examined quantitatively in an orchard. Lycopodium spores were utilized because they could be purchased from a commercial source and liberated in known amounts. The results of this study were compared to results of other studies completed within and above field crops to determine if spore dispersal patterns were different in an orchard and if previously developed spore dispersal models could be used to describe the dispersal of V. inaequalis ascospores. In addition, an attempt was made to relate airborne ascospore density to lesion density on McIntosh trees and to determine the potential effect of inoculum originating outside an orchard on the development of scab epidemics within the orchard. Inoculum originating outside an orchard could be important in New Hampshire where many abandoned or untended apple trees dot the countryside.

## Materials and Methods

All spore dispersal studies were conducted in a 0.5 ha block of semi-dwarf McIntosh apple trees on EM-7 rootstock, approximately 20 years old, at the University of New Hampshire Woodman Farm. The orchard was chosen because it was 1.6 km from the nearest apple orchard and thus relatively isolated from outside inoculum sources. The spore traps used were designed by Zuck at the University of Maine (135,136; Appendix A). These traps were chosen because they were relatively inexpensive to build, easy to use, and volumetric.

**Ascospore dispersal.** The dispersal of V. inaequalis ascospores was

studied during the primary apple scab season in 1982 and 1983. Placement of the traps in the orchard was determined after examining meteorological data (courtesy of Pease AFB, Newington, NH) to determine prevailing wind directions during spring rains. Wind direction varied but was most frequently from the NE to SE. Eighteen spore traps were utilized in 1982 (Fig. 1), and 21 traps were used in 1983 (Fig. 2).

A point source of inoculum was utilized both years. The inoculum consisted of scabbed leaves picked from unsprayed McIntosh trees at the Mast Road research orchard. The leaves were overwintered in wire cages and then transferred to the Woodman Farm orchard in April, prior to the detection of the first mature ascospore. The cages, arranged in a circle, contained 1000 leaves in 1982 and 1700 leaves in 1983.

To minimize background inoculum and to ensure that most ascospores trapped came from the point source, all leaves in the orchard were raked and removed in November of 1981. Although less than 0.01% of the leaves in the orchard had scab in Sept, 1982, a post-harvest application of benomyl (0.9 kg ai $ha^{-1}$) was applied in Nov, 1982 to reduce the amount of overwintering inoculum in the orchard.

Ascospores were released naturally during rainy periods. The spore traps were turned on and off automatically during rainy intervals using the system described in Appendix B. Temperature ($^{o}F$), hours of leaf wetness and hours of trap operation, relative humidity (%) and rainfall (hundreths mm) were recorded with instrumentation developed at the University of New Hampshire (81). Wind speed and direction were measured with a recording anemometer (Model W200-SD,

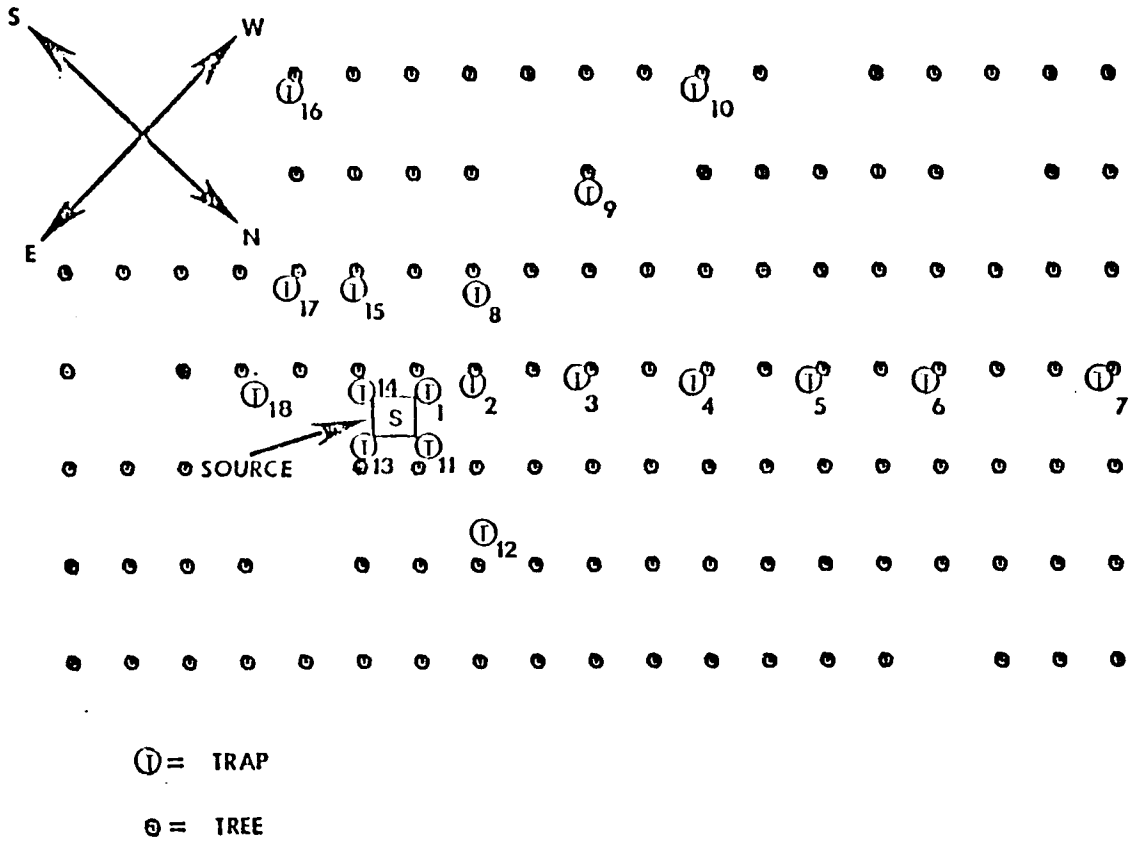



Figure 1. Trap placement, design A, for studying ascospore dispersal in the spring, 1982. Source, (s) contained approximately 1000 scabbed leaves in a 4 $m^2$ area.

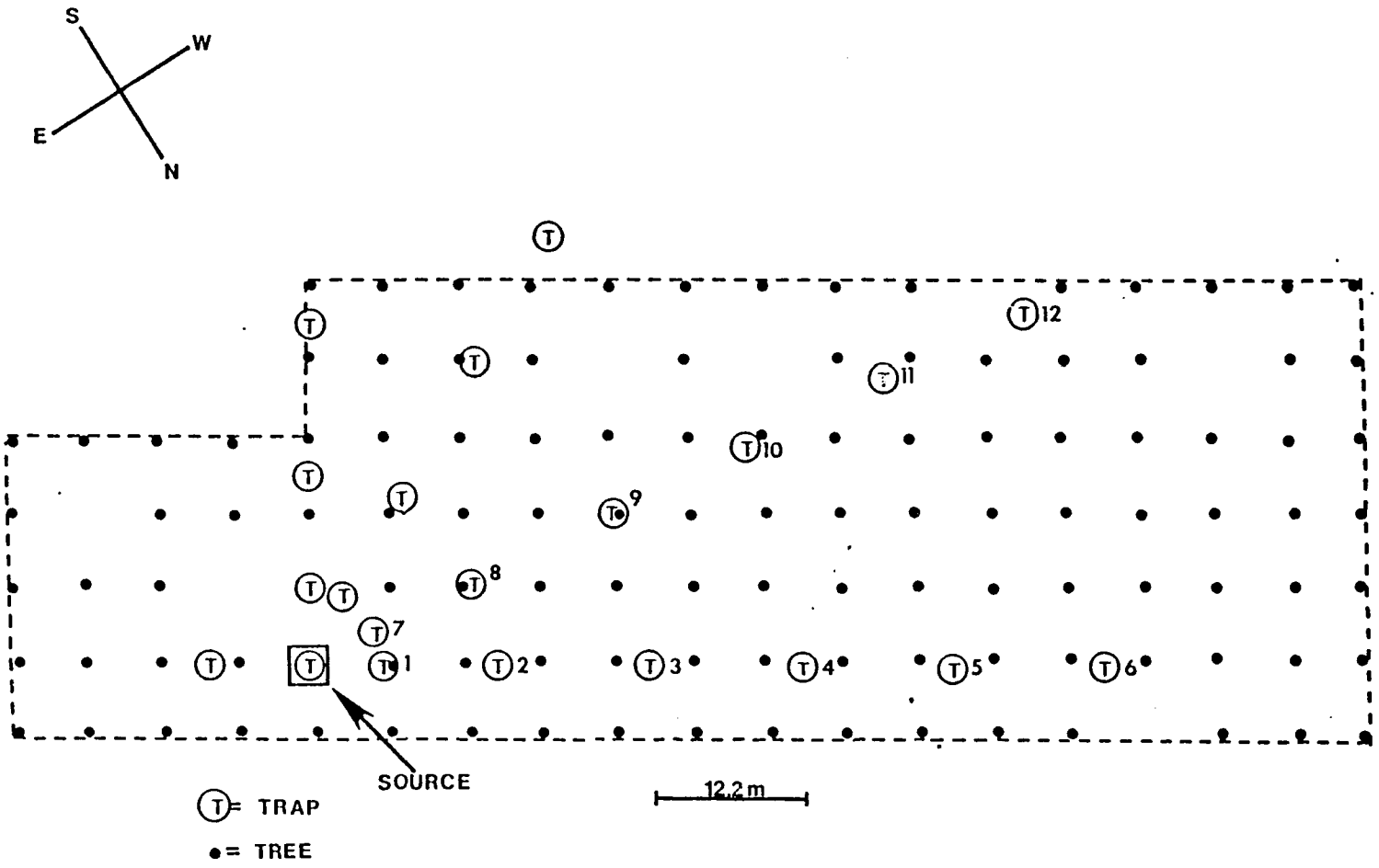



Figure 2. Trap placement, design B, for studying ascospore and Lycopodium spore dispersal in the spring, 1983. The ascospore source contained approximately 1700 leaves in a 4 $m^2$ area. Lycopodium spores (0.5 g) were released from a point just below the spore trap place at the point source.

Weather Measure Corp., Sacramento, CA 95841).

Attempts were also made to relate airborne ascospore density to lesion density. During each fungicide application throughout the primary scab season in 1982 and 1983, plastic trash bags were placed over 10 limbs in the immediate vicinity of each spore trap to provide unprotected tissue for each primary infection period. At the end of the primary season, cluster leaves, terminal leaves, and fruit on these limbs were indexed for scab incidence.

**Lycopodium dispersal.** The dispersal of spores of Lycopodium sp was studied in 1983 and 1984 utilizing four experimental designs. The first (Design C, Fig. 3) examined the variation in direction of spore dispersal over time, and consisted of 22 spore traps at 0.9 m above ground level arranged in a 4 m diameter circle. Twelve grams of a commercial preparation of Lycopodium spores (Carolina Biological Supply Co, Burlington, NC 27215) were released from the center of the circle in 1 gram aliquots at 15 min intervals for 3 hr. Spores were released for 30 s and the traps were left on for 2 min. The glass slides in the spore traps were changed after every four releases to avoid an uncountable build-up of Lycopodium spores. The slides were coated with Haupt's fixative (Carolina Biological Supply Co) to increase the impaction efficiency of the Lycopodium spores. The Lycopodium spores were released using an apparatus described in Figure 4. Each release period lasted 30 s, but most spores were released in the first 10 s of each release.

The second experiment examined horizontal dispersal within the orchard up to 64 m from the source. Placement of the traps is depicted

in Figure 2. For these tests, 0.5 g of Lycopodium spores were released from a point 1 m upwind of the source trap. Spores were released for 30 s and the spore traps were operational for 2 min.

A third study investigated horizontal dispersal up to 26 m (Design D, Fig. 5). Lycopodium spores (0.5 g) were released as described in the preceding test. Similar tests in 1984 investigated dispersal of Lycopodium spores up to 30 m from the source (Design E, Fig. 6), but the amount of spores released per test was increased from 0.5 to 5.0 g. In each test, the spores were released for 2 min and the spore traps were in operation for 5 min.

In all V. inaequalis and Lycopodium spore tests, the deposited spores were mounted in Gelvatol:glycerol:water (35 g:100 ml:50ml). The entire band of deposited spores was examined, and the number of spores recorded as "spores per slide" and spores $m^{-3}$ air.

**Statistical Analysis.** Dispersal data was analyzed with multiple regression, using the general equation,

$$Y = a + bX_1 + cX_1^2 + dX_1^3 + eX_2,$$

where y is the log of the number of spores trapped, $X_1$ is the log of the distance from the source, and $X_2$ is a constant between 0 and 3 to relate trap location to the direction of the wind, with 0 being directly downwind from the source, and 1, 2, and 3, being respectively, 30, 60, and 90$^{o}$ off the wind.

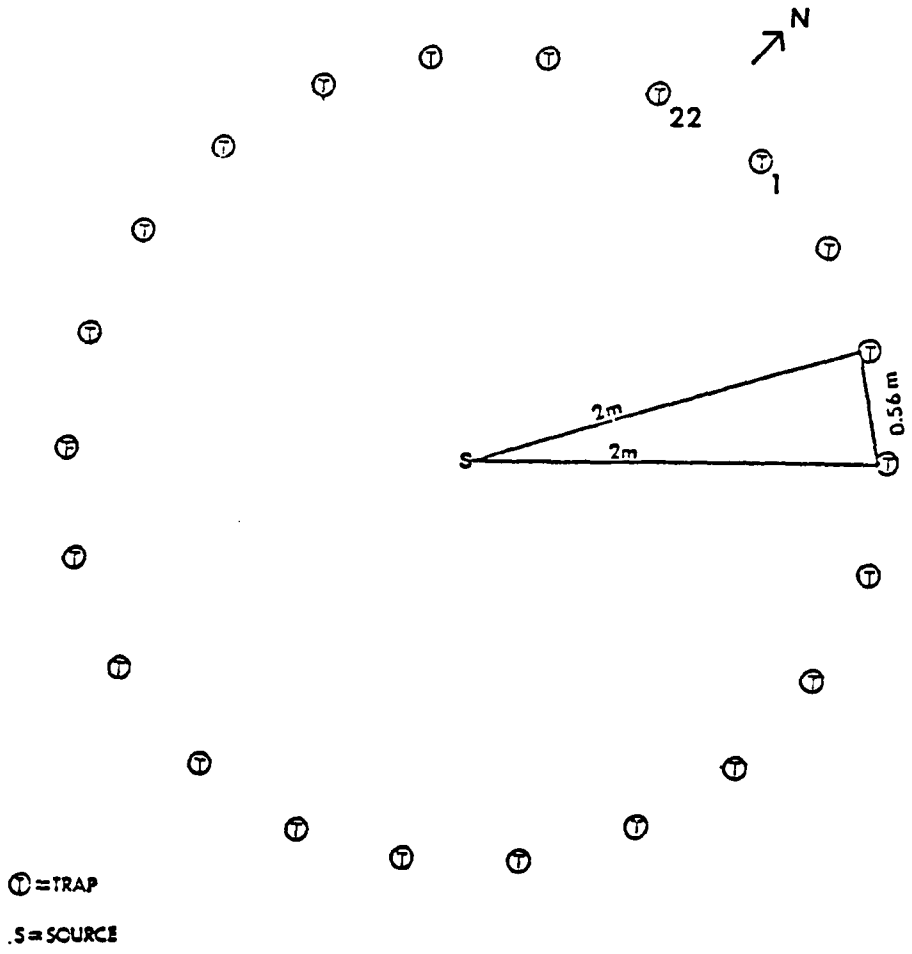



Figure 3. Trap placement, design C, for studying Lycopodium spore dispersal. One g of Lycopodium spores was released from the center of the circle every 15 min for 3 h.

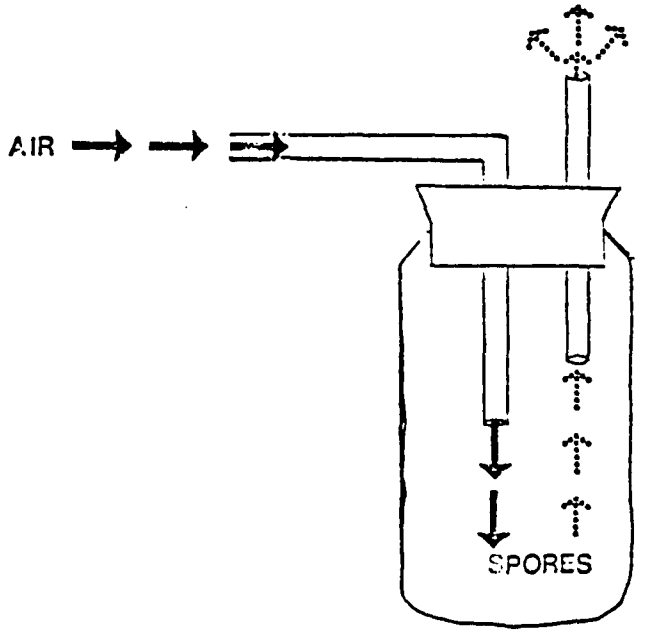



Figure 4. Apparatus for releasing Lycopodium spores. Spores were placed in a wide mouth jar sealed with a rubber stopper. Air was forced into the jar from the output of a laboratory vacuum cleaner. Spores were lifted from the bottom of the jar and carried by a stream of air through a glass tube into the environment.

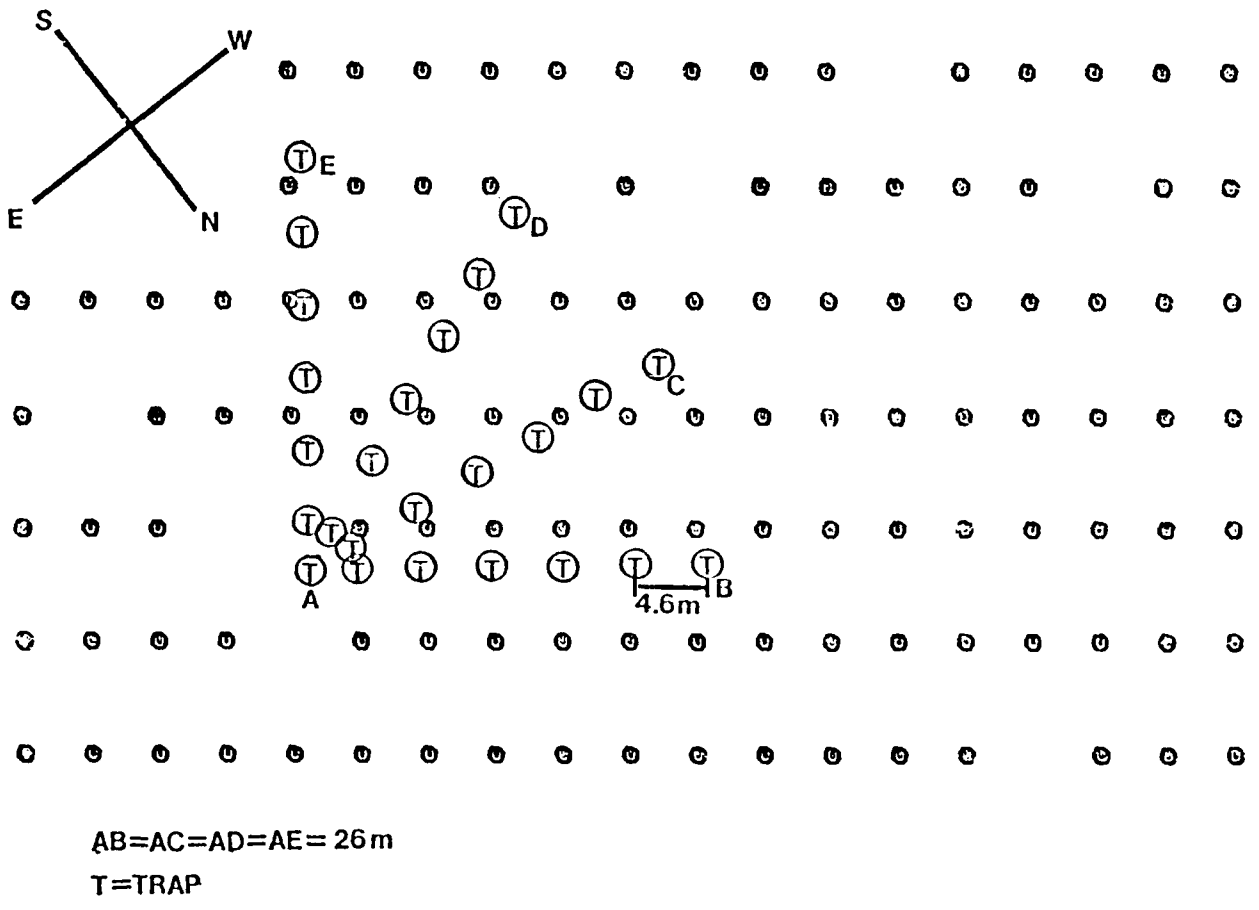



Figure 5. Trap placement, design D, for studying Lycopodium dispersal. For each test, 0.5 g of Lycopodium spores were released from a point source (A).

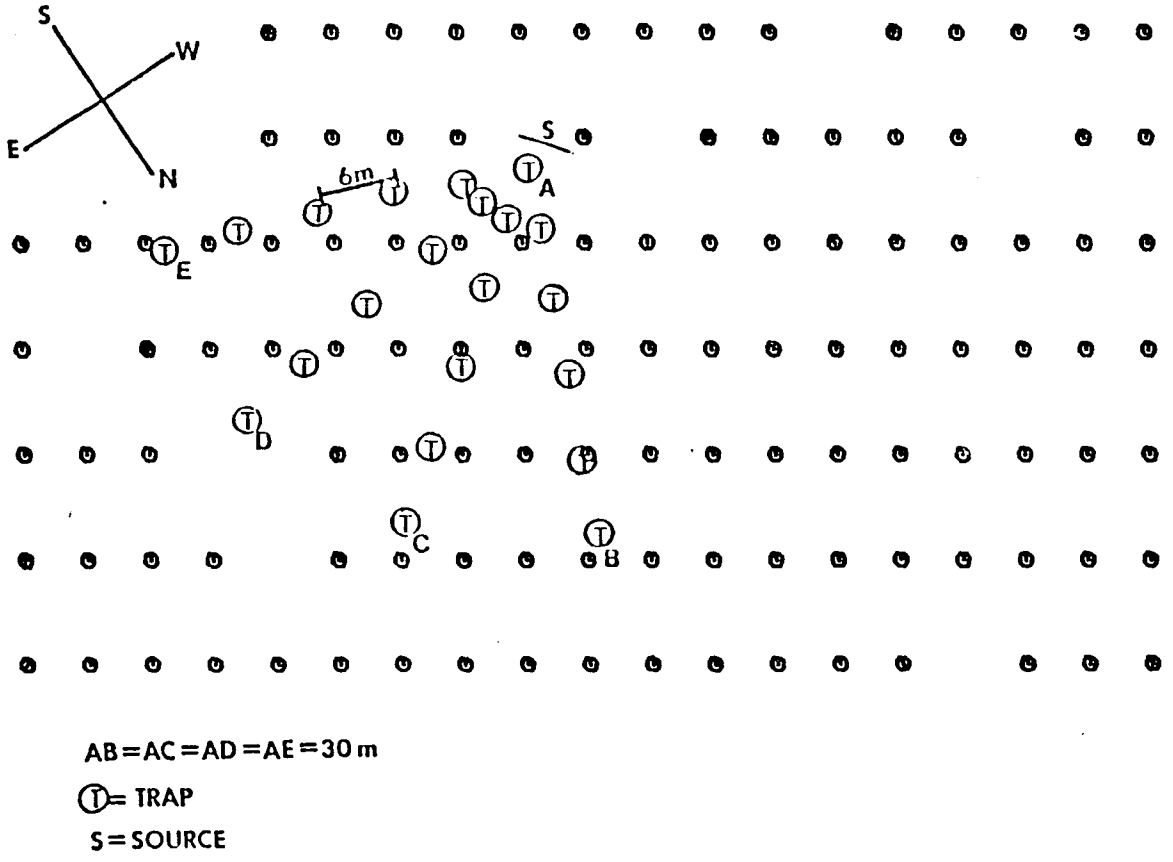



Figure 6. Trap placement, design E, for studying Lycopodium spore dispersal. For each test, 5.0 g of Lycopodium spores were released from a point (S) 1.0 m upwind of point A.

# Results

## Ascospore dispersal

**1982.** With the spore traps placed according to Design A (Fig. 1), dispersal was studied during four infection periods, with at least one line of spore traps downwind of the inoculum source during three of the infection periods. Only two spore traps were downwind on 19-20 May, when the wind was from the W and SW.

Few ascospores were trapped during each infection period, with most usually trapped closest to the inoculum source. Because few ascospores were trapped, a spore density gradient was usually not detected. The steepest gradients were recorded on 23-25 May during the largest ascospore release of the year. Gradients were less steep or not apparent during other releases. Beyond 15 m, there was no discernable gradient of ascospores trapped (Fig. 7). Along the longest transect (Traps 2-7), the number of ascospores trapped 76 m from the source was often equal to or greater than the number of spores trapped 21 or 33 m from the source. The only significant ($p = 0.05$) regression was calculated when the data for all the releases were combined (Table 1).

**1983.** In 1983, the weather was not favorable to study ascospore dispersal with the experimental design (Design B, Fig. 2). There were 10 daytime rains with mature ascospores available for discharge, but only on 24-25 April and 11 May did the wind blow from a direction

favorable for spore dispersal in the direction of the traps (E to ESE on 24-25 April and NE-SE, variable, on 11 May). During the other rainy periods, wind direction was N, W or SW, leaving the traps upwind of the inoculum source.

The infection period on 24-25 April allowed four sampling periods, but the brief period on 11 May allowed only one sampling period. Regression analysis of each sampling period did not produce any statistically significant equations, but a significant ($p = 0.05$) regression equation was generated when all sampling periods were combined (Table 1). The patterns of ascospore dispersal for the two periods are depicted in Figures 8a and 8b.

**Disease assessment.** In 1982 and 1983, all limbs covered with plastic bags during fungicide application were indexed two weeks after the conclusion of the primary season. The average number of clusters and terminals at each site was 85 and 69, respectivley. The data from both years are presented in Tables 2 (1982) and 3 (1983).

Few lesions developed each year. Twenty-five cluster lesions were recorded in 1982, 15 of them at two sites 3.0 m from the inoculum source. No other site had more than two lesions and ten sites had no cluster lesions. In 1983, there were only five cluster lesions at 12 sites, with three lesions occurring at one site 15 m from the source. Forty-one and 67% of the sites had no terminal lesions in 1982 and 1983, respectively. In 1982, 10 of 17 lesions occurred 9 m or less from the source, and 8 of 13 terminal lesions in 1983 occurred at one site 6.0 m from the source. Regression analysis could not determine a statistically significant relationship between lesion numbers and distance from the source.

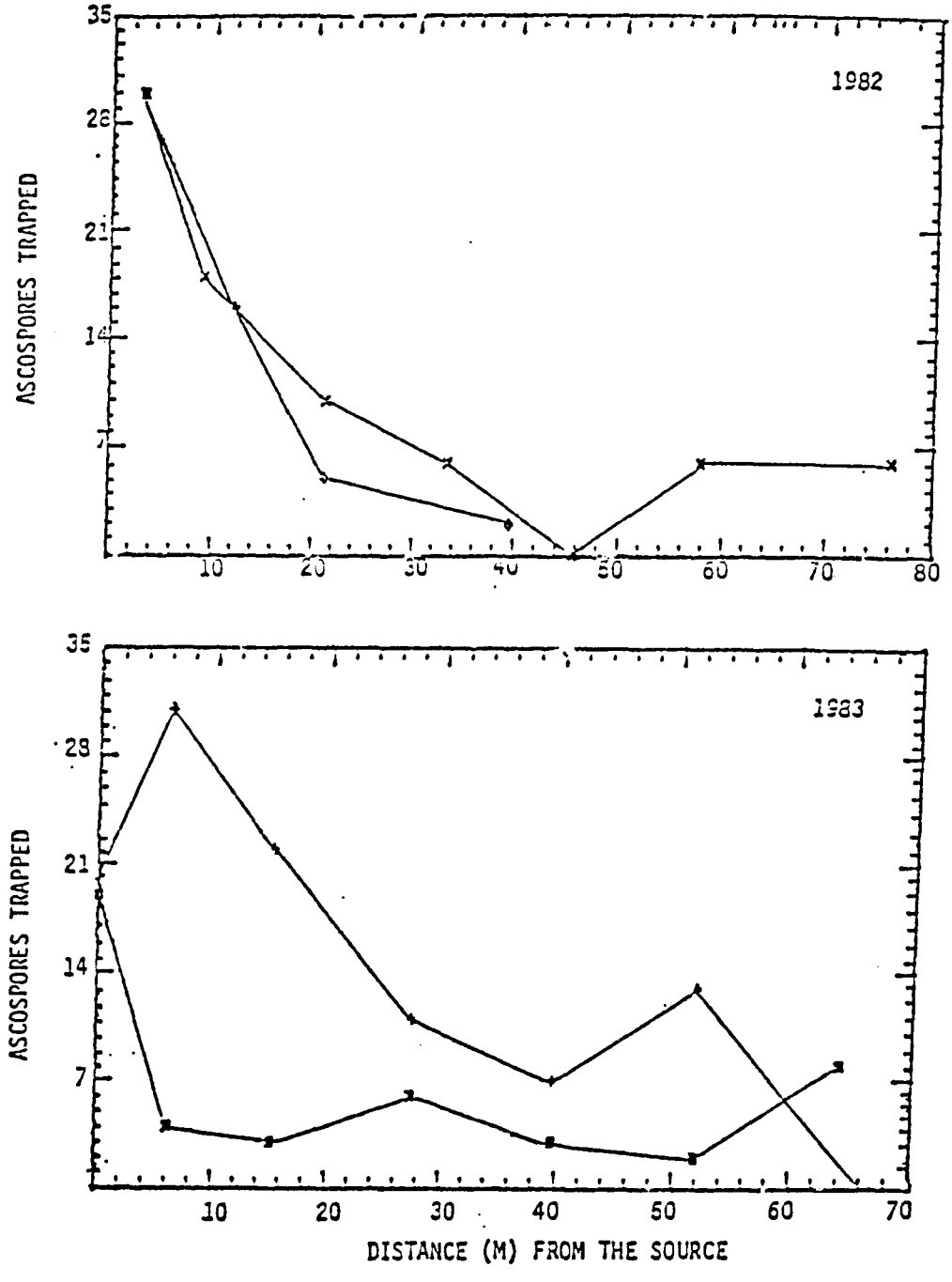



Figure 7. Typical pattern of ascospore dispersal along two transects with spore traps located downwind of an inoculum source containing approximately 1000 (1982) or 1700 (1983) scabbed leaves. Each curve represents a dispersal pattern for one infection period.

Table 1. Regression equations for ascospore dispersal, 1982 and 1983. The general equation is $Y = a + bX_1 + cX_1^2 + dX_1^3 + eX_2$, where Y = log of ascospores trapped, $X_1$ is log of distance from the inoculum source, and $X_2$ is a factor to relate trap location to the mean wind direction, with a value of 0 when traps were directly downwind, and 1,2,and 3 when traps were 30, 60 and 90° off the wind, respectively. All regressions were tested at the 95% confidence level; ns = not significant.

| Test | Date | a | b | c | d | e | $R^2$ | Standard Error |
|---|---|---|---|---|---|---|---|---|
| | 1982 | | | | | | | |
| 1 | 9-10 May | ns | ns | ns | ns | ns | .38 | |
| 2 | 19-20 May | ns | ns | ns | ns | ns | .31 | |
| 3 | 23-25 May | ns | ns | ns | ns | ns | .25 | |
| 4 | 29 May - 2 June | ns | ns | ns | ns | ns | .37 | |
| 1982 Total | | 1.78 | ns | -0.859 | 0.342 | ns | .56 | ± 0.0494 |
| | 1983 | | | | | | | |
| 1 | 24 April | ns | ns | ns | ns | ns | .13 | |
| 2 | 25 April | ns | ns | ns | ns | ns | .14 | |
| 3 | 25 April | ns | ns | ns | ns | ns | .28 | |
| 4 | 25-26 April | ns | ns | ns | ns | ns | .32 | |
| 5 | 11 May | ns | ns | ns | ns | ns | .44 | |
| 1983 Total | | 2.02 | -1.27 | 0.541 | ns | ns | .46 | ± 0.0520 |

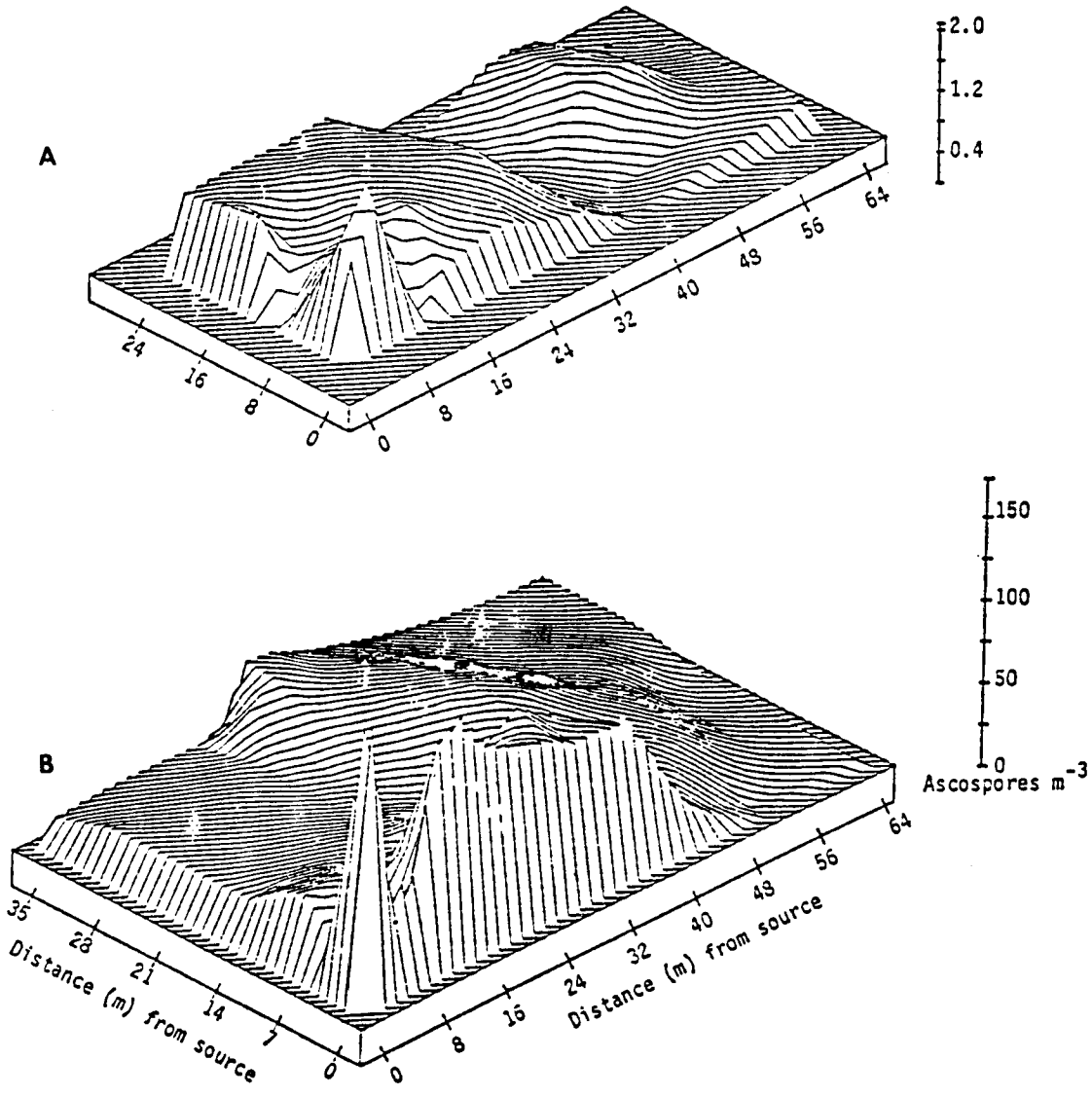



Figure 8. Patterns of ascospore dispersal on 24-25 April (A) and 11 May (B), 1983. Spore traps were set up according to design B (Fig. 2).

Table 2. Lesions recorded and ascospores trapped at specified distances from a point source of inoculum. Leaves were unprotected by fungicide during the 1982 primary apple scab season.

| Site[1] | Distance (m) from inoculum source | Lesions[2] | | Ascospores[3] trapped |
|---|---|---|---|---|
| | | Cluster | Terminal | |
| 1 | 3.0 | 7 | 1 | 47 |
| 2 | 9.1 | 2 | 0 | 31 |
| 3 | 21.3 | 2 | 3 | 16 |
| 4 | 33.5 | 0 | 0 | 14 |
| 5 | 44.7 | 0 | 0 | 6 |
| 6 | 57.9 | 2 | 0 | 12 |
| 7 | 76.1 | 1 | 1 | 12 |
| 8 | 12.2 | 1 | 0 | 36 |
| 9 | 21.3 | 0 | 0 | 12 |
| 10 | 39.5 | 0 | 1 | 7 |
| 11 | 3.0 | 0 | 0 | 39 |
| 12 | 12.2 | 2 | 0 | 11 |
| 13 | 3.0 | 0 | 0 | 27 |
| 14 | 3.0 | 8 | 5 | 29 |
| 15 | 9.1 | 0 | 4 | 20 |
| 16 | 21.3 | 0 | 2 | 4 |
| 17 | 12.2 | 0 | 0 | 10 |
| 18 | 15.3 | 0 | 0 | 12 |

[1]Site locations presented in Fig. 1.

[2]Number of lesions recorded for a mean of 85 clusters and 69 terminals at each site.

[3]Total ascospores trapped during the primary season by spore trap placed at each site.

Table 3. Lesions recorded and ascospores trapped at specified distances from a point source of inoculum. Leaves were unprotected by fungicide during the 1983 primary apple scab season.

| Site[1] | Distance (m) from inoculum source | Lesions[2] | | Ascospores trapped[3] |
|---|---|---|---|---|
| | | Cluster | Terminal | |
| 1 | 6.1 | 0 | 8 | 26 |
| 2 | 15.2 | 3 | 0 | 15 |
| 3 | 27.4 | 1 | 0 | 8 |
| 4 | 39.6 | 0 | 1 | 16 |
| 5 | 51.8 | 0 | 2 | 13 |
| 6 | 64.0 | 0 | 0 | 10 |
| 7 | 6.1 | 0 | 0 | 42 |
| 8 | 15.2 | 0 | 0 | 31 |
| 9 | 27.4 | 0 | 0 | 20 |
| 10 | 39.6 | 1 | 0 | 14 |
| 11 | 51.8 | 0 | 0 | 19 |
| 12 | 64.0 | 0 | 2 | 21 |

[1]Site locations presented in Fig. 2.

[2]Number of lesions recorded for a mean of 85 clusters and 69 terminals at each site.

[3]Total ascospores trapped during the primary season by spore trap placed at each site.

Lycopodium studies

**Design C (Fig. 3).** Four tests were completed using the circular design. Weather conditions for all tests were dry, although cloud cover varied from none to cloudy. Wind velocity was generally light, with a maximum of 4.8 m $s^{-1}$ on 31 Oct. Wind velocity and direction for each test are given in Table 4.

When spore trapping data for all releases comprising a test were combined, at least 1 spore was trapped in every trap, but spores were not always recorded at each trap for individual spore releases (Table 5). There was a 150 fold (15 Nov) to 12,650 fold (29 Nov) difference in the number of spores trapped upwind and downwind from the source. The directional range of spore dispersal was equal to or greater than the range in wind direction (Appendix C, Figs. 1-4). More spores were trapped when winds were lighter (Table 4). From visual observations of spore cloud dispersal made during the tests, it was determined that in stronger winds spores tended to remain close to the ground near the source, with less vertical dispersion. With lighter winds, vertical dispersal was greater closer to the source and the center of the spore cloud was more closely aligned with the spore traps which were at a height of 0.9 m.

**Horizontal Dispersal: Design B (Fig. 2).** Eight tests were conducted when the wind direction was from the SE or E, thereby insuring that at least one line of six spore traps was downwind of the spore source. Few spores were trapped during any test (Table 6; Appendix C, Fig. 5a-g). Excluding the trap placed directly above the release point (S),

the greatest number of spores was trapped by the spore trap closest (6.1 m) to the spore source. One exception was test 4 (Table 6), when the most spores were trapped 52 m from the source. Only in three instances were more than ten spores (110 spores $m^{-3}$ air) trapped (Table 6), once in test four at trap 11, and twice in test 6 at traps one and eleven. Significant regression equations were calculated for tests two, three and six (Table 7), but the regression in test two is misleading because there were seven traps which trapped no spores (Table 6). A significant regression was also calculated when the data from all seven tests were combined (Table 7). The dispersal patterns for the tests, presented in Appendix C (Fig. 6a-g), were similar to the ascospore dispersal patterns presented in Figure 8.

**Horizontal Dispersal: Design D (Fig 5).** Six tests were completed on 22 August, 1983 (Table 8), a clear day with wind from the S, but varying to E to S x W. Wind velocity for the first three tests was moderate, from 0.9 - 5.3 m $s^{-1}$, then less (0.0 - 1.8 m $s^{-1}$) for the remaining tests.

In each test, the most spores were trapped at one of the four spore traps closest to the spore source (Table 8; Appendix C, Fig. 7a-f). Comparing the maximum number of spores trapped by any trap to the number trapped 0.9 m above the inoculum source, there were always fewer trapped above the source. The most spores trapped by the spore trap placed above the source were from releases during light wind conditions. The number of spores trapped at each site in tests 1-4 were similar to those trapped during Lycopodium tests using design A. Tests five and six had more spores trapped than during any test using

Table 4. The effect of wind velocity on the number of spores of Lycopodium sp trapped by 22 "Zuck" volumetric spore traps arranged in a circle with a diameter of 4 m (design C, Fig. 3). Each test lasted three hours, with 1.0 g of spores released every 15 minutes.

| Test Date | Weather conditions | Wind parameters: Directional range (°) | Velocity ($m\ s^{-1}$) Range | Velocity ($m\ s^{-1}$) Mean | Airborne spore density (spores $m^{-3}$ air)[1] |
|---|---|---|---|---|---|
| 31 Oct | Clear, 14 C | 40 | 0-4.8 | 1.99 | 155,896 |
| 1 Nov | Clear, 11 C | 190 | 0-3.1 | 1.39 | 188,489 |
| 27 Nov | Cloudy, 10 C | 55 | 0-2.7 | 0.99 | 221,510 |
| 15 Nov | Clear, -2 C | 80 | 0-1.8 | 0.52 | 295,691 |

[1]Total spores trapped by all traps.

Table 5. Lycopodium spore densities (spores $m^{-3}$ air) at each site for tests completed with spore traps aligned in a circular pattern around a point source of spores (design C, Fig. 3). Releases A, B, and C included the spores trapped in the first, second, and third hour of each test, respectively. During each hour, 4.0 g of Lycopodium spores were released, 1.0 g released every 15 min.

| Test | Release | Trap 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | A | 29.4 | 44.1 | 7.3 | 14.7 | 36.7 | 51.5 | 441.1 | 4750.0 | 1654.4 | 5080.8 | 1500.0 |
| | B | 36.7 | 22.0 | 0 | 7.3 | 2507.3 | 4272.0 | 3433.3 | 5933.8 | 20220.5 | 18757.3 | 16448.5 |
| | C | 112.5 | 6779.4 | 10338.2 | 21250.0 | 47426.4 | 5676.4 | 53375.0 | 24066.1 | 51102.9 | 45875.0 | 23735.2 |
| | Total | 59.5 | 2281.8 | 3448.5 | 7090.6 | 16656.8 | 3333.2 | 19083.1 | 11583.3 | 24325.9 | 23237.7 | 13894.5 |
| 2 | A | 1279.4 | 12220.5 | 9330.8 | 2073.5 | 2389.7 | 3911.7 | 2838.2 | 95.6 | 7.3 | 7.3 | 29.4 |
| | B | 8147.0 | 6367.6 | 1036.7 | 66.2 | 0 | 36.7 | 14.7 | 0 | 7.3 | 14.7 | 22.0 |
| | C | 8617.0 | 19514.7 | 17375.0 | 56014.7 | 27338.2 | 14.7 | 7.3 | 117.6 | 0 | 29.4 | 22.0 |
| | Total | 6014.4 | 9034.3 | 9247.5 | 19384.8 | 9909.3 | 1321.0 | 953.4 | 71.1 | 4.8 | 17.1 | 24.4 |
| 3 | A | 19757.3 | 3610.3 | 5750.0 | 7852.9 | 5580.8 | 75779.4 | 79801.4 | 63625.0 | 125.0 | 7.3 | 125.0 |
| | B | 9602.9 | 4713.2 | 1044.1 | 3691.1 | 21735.3 | 29220.6 | 43250.0 | 4573.5 | 19213.2 | 75948.5 | 30500.0 |
| | C | 8504.5 | 26360.3 | 60558.5 | 16000.0 | 4135.1 | 78810.8 | 34648.6 | 1774.7 | 90.1 | 252.2 | 126.1 |
| | Total | 13200.5 | 10850.3 | 20443.8 | 8946.5 | 11160.4 | 61572.2 | 55029.4 | 25326.2 | 7058.8 | 27695.2 | 11173.8 |
| 4 | A | 16088.2 | 2610.3 | 32316.1 | 49036.7 | 34183.8 | 45705.8 | 42301.5 | 12661.7 | 6198.5 | 970.6 | 22.1 |
| | B | 742.6 | 779.4 | 11441.1 | 17294.1 | 34713.2 | 31007.3 | 33691.1 | 2823.5 | 13911.7 | 12772.1 | 4845.6 |
| | C | 2352.9 | 2529.4 | 29000.0 | 31147.0 | 45691.1 | 2985.3 | 2926.4 | 124897.0 | 882.3 | 44.1 | 0 |
| | Total | 7202.9 | 1861.7 | 23302.9 | 32761.7 | 36697.0 | 30685.3 | 30982.3 | 31173.5 | 8220.6 | 5505.8 | 1950.0 |

Table 5. cont.

| Test | Release | Trap 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | A | 5558.8 | 5080.8 | 2397.0 | 0 | 44.1 | 0 | 22.0 | 0 | 22.0 | 14.7 | 36.7 |
| | B | 27897.0 | 27522.0 | 51.4 | 102.9 | 22.0 | 14.7 | 7.3 | 0 | 0 | 29.4 | 29.4 |
| | C | 16485.3 | 12529.4 | 911.7 | 66.2 | 6720.5 | 3985.3 | 794.1 | 2838.2 | 66.2 | 566.2 | 235.3 |
| | Total | 16647.0 | 15044.0 | 1120.0 | 56.3 | 2262.2 | 1333.3 | 274.4 | 946.0 | 29.4 | 203.4 | 100.4 |
| 2 | A | 36.7 | 66.2 | 595.5 | 1588.2 | 94132.3 | 11014.7 | 52823.5 | 28698.5 | 17301.4 | 19433.8 | 10198.5 |
| | B | 22.0 | 29.4 | 0 | 44.1 | 2566.1 | 2360.3 | 904.4 | 2176.4 | 14264.7 | 23602.9 | 33727.9 |
| | C | 14.7 | 29.4 | 7.3 | 0 | 95.5 | 176.4 | 926.4 | 8963.2 | 37720.5 | 19161.7 | 20732.8 |
| | Total | 24.4 | 41.6 | 200.9 | 544.1 | 32264.6 | 4517.1 | 18218.1 | 13279.3 | 23095.5 | 20732.8 | 19588.2 |
| 3 | A | 7.3 | 14.6 | 14.6 | 29.4 | 205.8 | 73.5 | 7.3 | 235.3 | 9830.8 | 11227.9 | 16088.2 |
| | B | 3669.1 | 6014.7 | 26867.6 | 13330.8 | 10102.9 | 19323.5 | 8411.7 | 889.7 | 88.2 | 51.5 | 9786.7 |
| | C | 36.0 | 45.0 | 27.0 | 27.0 | 270.2 | 27.0 | 54.0 | 0 | 6486.5 | 7423.4 | 11585.5 |
| | Total | 1347.6 | 2205.8 | 9783.4 | 4866.3 | 3828.8 | 7061.5 | 3077.5 | 409.1 | 5532.1 | 6304.8 | 12847.6 |
| 4 | A | 0 | 691.2 | 264.7 | 2757.3 | 2610.3 | 1051.5 | 1323.5 | 338.2 | 235.3 | 1205.8 | 4823.5 |
| | B | 7.3 | 14.6 | 7.3 | 7.3 | 110.3 | 0 | 7.3 | 0 | 14.6 | 22.0 | 220.6 |
| | C | 0 | 0 | 29.4 | 0 | 44.1 | 14.7 | 29.4 | 29.4 | 14.7 | 14.7 | 58.8 |
| | Total | 2.9 | 282.3 | 108.8 | 1105.8 | 1105.8 | 423.5 | 538.2 | 141.2 | 102.9 | 494.1 | 2029.4 |

Table 6. Airborne spore densities (spores $m^{-3}$ air) for Lycopodium releases with spore traps aligned along horizontal transects from a point source of spores (design B, Fig. 2).

| Trap | Distance (m) from source | Test 1 | 2 | 3 | 4 | 5 | 6 | 7 | Total[1] |
|---|---|---|---|---|---|---|---|---|---|
| S | 0.0 | 126 | 116 | 116 | 105 | 316 | 2063 | 253 | 3096 |
| 1 | 6.1 | 95 | 53 | 95 | 21 | 42 | 168 | 11 | 484 |
| 2 | 15.2 | 11 | 0 | 33 | 22 | 0 | 56 | 0 | 122 |
| 3 | 27.4 | 11 | 22 | 11 | 22 | 22 | 22 | 11 | 122 |
| 4 | 39.6 | 33 | 44 | 33 | 33 | 11 | 11 | 11 | 178 |
| 5 | 51.8 | 55 | 0 | 22 | 56 | 22 | 11 | 22 | 189 |
| 6 | 64.0 | 21 | 0 | 42 | 21 | 32 | 11 | 11 | 137 |
| 7 | 6.1 | 11 | 11 | 21 | 84 | 32 | 11 | 21 | 189 |
| 8 | 15.2 | 11 | 0 | 22 | 56 | 22 | 11 | 0 | 122 |
| 9 | 27.4 | 22 | 0 | 22 | 89 | 22 | 11 | 0 | 167 |
| 10 | 39.6 | 11 | 0 | 11 | 44 | 11 | 0 | 0 | 78 |
| 11 | 51.8 | 0 | 0 | 12 | 129 | 36 | 12 | 0 | 188 |
| 12 | 64.0 | 33 | 0 | 22 | 56 | 22 | 11 | 0 | 144 |
| 13 | 6.1 | 10 | 10 | 20 | 40 | 20 | 190 | 30 | 320 |
| 14 | 15.2 | 10 | 0 | 10 | 10 | 20 | 40 | 40 | 130 |
| 15 | 27.4 | 20 | 0 | 20 | 10 | 0 | 10 | 10 | 70 |
| 16 | 39.6 | 10 | 20 | 0 | 0 | 20 | 10 | 10 | 70 |
| 17 | 6.1 | 53 | 11 | 42 | 21 | 42 | 53 | 11 | 232 |
| 18 | 15.2 | 20 | 0 | 40 | 40 | 20 | 20 | 10 | 150 |
| 19 | 27.4 | 21 | 31 | 53 | 31 | 42 | 11 | 11 | 199 |

[1]Total spores trapped by each trap for all tests.

Table 7. Regression equations for Lycopodium spore dispersal. The general equation, $Y = a + bX_1 + cX_1^2 + dX_1^3 + eX_2$ was used, where Y = log of spores trapped, $X_1$ is log of distance from the spore source and $X_2$ is a factor to relate trap location to mean wind direction, with a value of 0 when traps were directly downwind and 1, 2, or 3 when traps were 30, 60, or $90^{o}$ off the wind, respectively. All regressions were tested at the 95% confidence level; ns = not significant.

| Test | a | b | c | d | e | $R^2$ | Standard Error |
|---|---|---|---|---|---|---|---|
| Design B | | | | | | | |
| 1 | ns | ns | ns | ns | ns | .06 | |
| 2 | 102 | -257 | 202 | -51.2 | ns | .55 | ± 2.565 |
| 3 | 1.12 | -0.737 | ns | ns | 0.961 | .91 | ± 0.1023 |
| 4 | ns | ns | ns | ns | ns | .14 | |
| 5 | ns | ns | ns | ns | ns | .19 | |
| 6 | -0.935 | ns | ns | ns | 1.19 | .22 | ± 1.669 |
| 7 | ns | ns | ns | ns | ns | .16 | |
| Total | 2.81 | -2.4 | 0.816 | ns | ns | .51 | ± 0.0225 |
| Design D | | | | | | | |
| 1 | 0.946 | -2.53 | ns | ns | 1.41 | .31 | ± 2.933 |
| 2 | ns | ns | ns | ns | ns | .11 | |
| 3 | ns | ns | ns | ns | ns | .46 | |
| 4 | ns | ns | ns | ns | ns | .13 | |
| 5 | -0.718 | ns | ns | ns | 0.913 | .23 | ± 1.575 |
| 6 | ns | ns | ns | ns | ns | .06 | |
| Total | 2.36 | -0.757 | ns | ns | ns | .43 | ± 0.0814 |
| Design E | | | | | | | |
| 1 | 6.03 | -1.66 | ns | ns | -1.7 | .39 | ± 3.494 |
| 2 | 9.34 | -4.02 | ns | ns | -2.0 | .70 | ± 1.885 |
| 3 | 6.0 | -2.56 | ns | ns | -0.222 | .87 | ± 0.0752 |
| 4 | ns | ns | ns | ns | ns | .18 | |
| 5 | 5.84 | -2.52 | ns | ns | -0.228 | .69 | ± 0.223 |

Table 8. Lycopodium spore densities (spores $m^{-3}$ air) for tests completed with spore traps aligned along four horizontal transects from a point source of spores (design D, Fig. 5).

| Trap | Distance (m) from source | Test 1 | 2 | 3 | 4 | 5 | 6 | Total |
|---|---|---|---|---|---|---|---|---|
| 1 | 0.0 | 177.0 | 77.7 | 100.0 | 44.4 | 311.1 | 822.2 | 1532.4 |
| 2 | 3.0 | 111.1 | 33.3 | 122.2 | 11.1 | 177.7 | 1455.5 | 1910.9 |
| 3 | 7.6 | 144.4 | 11.1 | 55.5 | 33.3 | 344.4 | 955.5 | 1544.2 |
| 4 | 12.2 | 231.5 | 0 | 84.2 | 42.1 | 168.4 | 642.1 | 1168.3 |
| 5 | 16.8 | 222.1 | 10.5 | 94.7 | 21.0 | 347.4 | 347.4 | 1042.1 |
| 6 | 21.3 | 94.7 | 21.0 | 21.0 | 42.1 | 136.8 | 200.0 | 515.6 |
| 7 | 25.9 | 52.6 | 0 | 0 | 31.6 | 21.1 | 105.3 | 210.6 |
| 8 | 3.0 | 100.0 | 33.3 | 144.4 | 55.5 | 577.7 | 244.4 | 1155.3 |
| 9 | 7.6 | 44.4 | 33.3 | 111.1 | 55.5 | 99.9 | 277.7 | 621.9 |
| 10 | 12.2 | 77.7 | 33.3 | 77.7 | 99.9 | 11.1 | 111.1 | 410.8 |
| 11 | 16.8 | 0 | 21.1 | 73.7 | 31.6 | 10.5 | 105.3 | 241.6 |
| 12 | 21.3 | 0 | 10.5 | 31.6 | 52.6 | 10.5 | 84.2 | 189.4 |
| 13 | 25.9 | 0 | 31.6 | 21.1 | 31.6 | 21.1 | 0 | 105.4 |
| 14 | 3.0 | 22.2 | 111.1 | 44.4 | 311.1 | 11.1 | 11.1 | 511.0 |
| 15 | 7.6 | 55.5 | 77.7 | 55.5 | 222.2 | 0 | 22.2 | 433.1 |
| 16 | 12.2 | 0 | 0 | 66.6 | 33.3 | 22.2 | 0 | 122.1 |
| 17 | 16.8 | 10.5 | 31.5 | 42.1 | 315.7 | 10.5 | 10.5 | 420.8 |
| 18 | 21.3 | 10.5 | 21.1 | 21.1 | 200.0 | 21.1 | 21.1 | 294.9 |
| 19 | 25.9 | 10.5 | 42.1 | 73.7 | 21.0 | 10.5 | 21.1 | 178.9 |
| 20 | 3.0 | 333.3 | 77.7 | 133.3 | 400.0 | 11.1 | 33.3 | 988.7 |
| 21 | 7.6 | 22.2 | 33.3 | 11.1 | 255.5 | 22.2 | 88.8 | 433.1 |
| 22 | 12.2 | 22.2 | 22.2 | 0 | 22.2 | 44.4 | 11.1 | 122.1 |
| 23 | 16.8 | 10.5 | 21.0 | 10.5 | 21.0 | 0 | 157.9 | 220.9 |
| 24 | 21.3 | 0 | 31.6 | 0 | 0 | 10.5 | 94.7 | 136.8 |
| 25 | 25.9 | 10.5 | 10.5 | 0 | 42.1 | 10.5 | 126.3 | 199.9 |

design B. Regressions for each test and the combined data are presented in Table 7. Spore dispersal patterns for each test and the combined data are depicted in Appendix C (Fig. 8a-f).

**Horizontal Dispersal: Design E (Fig 6).** Design E was similar to design D, but the traps were placed at 6 m intervals up to 30 m from the source rather than 4.5 m intervals up to 23 m from the source. In each test, 5 g of Lycopodium spores were released, 10 times the number released in previous dispersal tests with Lycopodium spores.

The major effect of increasing the numbers of spores released was the detection of steep spore density gradients in each test. A maximum of 58,912 spores $m^{-3}$ air was measured 1 m from the source in test 3 (Table 9) while 99% fewer spores (76.4, 235.2, 229 and 53 spores $m^{-3}$ air) were trapped 30 m from the source in the same test. Similar gradients were detected in the other tests (Table 9; Appendix C, Fig. 9a-e). Significant regression equations were calculated for all tests except 4 in which the fewest spores were trapped (Table 7). The dispersal patterns for all tests are depicted in Appendix III (Fig 10a-e).

Table 9. *Lycopodium* spore densities (spores $m^{-3}$ air) for tests completed with spore traps aligned along four horizontal transects from a point source of spores (design E, Fig. 6).

| Trap | Distance (m) from source | Test 1 | 2 | 3 | 4 | 5 |
|---|---|---|---|---|---|---|
| 1 | 1 | 6958 | 37912 | 58912 | 3429 | 21758 |
| 2 | 6 | 1682 | 11894 | 7953 | 24 | 3906 |
| 3 | 12 | 700 | 8041 | 1447 | 29 | 147 |
| 4 | 18 | 271 | 4876 | 247 | 12 | 77 |
| 5 | 24 | 6 | 1500 | 94 | 0 | 18 |
| 6 | 30 | 6 | 965 | 76 | 6 | 59 |
| 7 | 6 | 1494 | 9547 | 4771 | 265 | 1759 |
| 8 | 12 | 812 | 2571 | 1088 | 53 | 1288 |
| 9 | 18 | 329 | 1812 | 853 | 59 | 506 |
| 10 | 24 | 682 | 1388 | 253 | 18 | 729 |
| 11 | 30 | 476 | 735 | 235 | 12 | 194 |
| 12 | 6 | 22835 | 5306 | 5512 | 5247 | 9418 |
| 13 | 12 | 6735 | 2035 | 2159 | 1941 | 2059 |
| 14 | 18 | 3994 | 6 | 1459 | 247 | 1018 |
| 15 | 24 | 794 | 12 | 82 | 706 | 547 |
| 16 | 30 | 194 | 6 | 229 | 706 | 147 |
| 17 | 6 | 2759 | 912 | 5018 | 4441 | 8853 |
| 18 | 12 | 100 | 6 | 377 | 347 | 500 |
| 19 | 18 | 0 | 0 | 341 | 129 | 153 |
| 20 | 24 | 0 | 12 | 47 | 377 | 35 |
| 21 | 30 | 6 | 0 | 53 | 18 | 35 |

## Discussion

One fact most apparent from this study is that successful modeling of spore dispersal requires either large numbers of spores or spore trapping over very short distances. An example of the latter is the study described in Chapter V of this dissertation. In the glasshouse, steep gradients of airborne ascospore density were detected within 2.8 m of an inoculum source of less than 200 scabbed leaves.

Ascospore dispersal in the orchard was entirely different from the glasshouse study, with no gradients detected for any infection period in 1982 or 1983. Ascospore densities increased and decreased randomly with distance from the inoculum source. Similar results were obtained for dispersal of Lycopodium spores when 0.5 g of spores were released. This pattern was observed when spore traps were placed up to 64 m (Design B) or 26 m from the source (Design D). One explanation for this pattern is that the presence of trees in the orchard increased turbulence, with air streams moving around trees. In the process, a spore cloud might by-pass one trap but pass by another further away. Another explanation is that air currents may move above trees at one location and then settle back into the orchard at another further downwind. This random pattern was different from those reported in other spore dispersal studies (9,47), but those studies had been conducted over field crops or grass fields, agroecosystems with much less air turbulence than an orchard.

Another possibility, however, is that the irregular patterns of dispersal were reflective of low spore densities away from the source

rather than complex wind currents. That is, rapid diffusion of the spore cloud resulted in airborne spore densities which were randomly dispersed beyond 10 m from the source. Airborne spore density gradients can not be detected when spores are randomly dispersed. The lack of correlation between airborne spore densities and distance using regression analysis in this study supports the contention that airborne spore numbers were too small to detect any predictable patterns.

By increasing the amount of spores released from 0.5 to 5.0 g in the Lycopodium tests, the calculation of statistically significant regression equations was possible for four tests. The one test with no correlation had the fewest number of spores trapped. The slope (b) of the regression lines for design E ranged from -1.66 to -4.02, with a mean of -2.69. Sreeramulu and Ramilingan (110) examined deposition of Lycopodium spores on glass slides placed horizontally up to 30 m from a point source. Values of b for their studies ranged from -1.174 to -2.119 (47). In the present study, b was higher, possibly because the volumetric spore traps used were much more efficient than glass slides in trapping spores and would trap a larger proportion of spores closer to the source resulting in a steeper gradient, given that flattening of the gradient was detected in both studies.

For the remaining Lycopodium tests, spore trapping data from each design were combined to calculate regression equations. The slopes for designs A and B were -2.4 and -0.757, respectively. Statistically significant regression equations were also calculated for ascospore dispersal in 1982 and 1983 by combining ascospore trapping data for

all release periods. While these equations predict a measurable gradient, the gradients are indicative of gradients that might occur if the season's supply of ascospores were released at once. A similar gradient from a single infection period would require three to five times the inoculum used in this study. The slopes of the lines were -0.859 (1982) and -1.27 (1983), indicating a less steep gradient for ascospores. The ascospore gradients were not a result of wind speed, as winds during infection periods were often as light as those measured during Lycopodium tests. Also, ascospore gradients were less steep despite the presence of rain during periods of ascospore release which would tend to reduce airborne ascospore density by a scrubbing-out process. That ascospore gradients were less steep than Lycopodium gradients is not surprising because the terminal velocity of ascospores is 0.2 $cm^{-2}$, compared to 1.76 - 2.14 $cm^{-2}$ for Lycopodium spores (46). Thus, more ascospores would remain in suspension at a given distance from a source than Lycopodium spores at the same distance, given an equal number released.

The inability to calculate statistically significant regression equations for individual ascospore release periods indicated that the distribution of ascospores was random within a short distance from the source. In 1982 and 1983, respectively, 85% and 60% of all ascospores trapped were within 30 m of the inoculum source. Beyond 30 m, the numbers of ascospores trapped varied irregularly with increasing distance from the source (Fig. 7). Thus, for this inoculum source of 1700 heaavily scabbed leaves, 30 m was sufficient for the gradient curve for airborne ascospores to flatten, and beyond this distance, the ascospores were randomly dispersed.

To relate this study to a situation that a commercial grower in New Hampshire might encounter, the following calculations were made to describe the inoculum source in terms of abandoned or untreated apple trees. Assume that all leaves on an unsprayed apple tree will fall beneath that tree and fill a circle with a diameter of 6.1 m (9.2 $m^2$). If the average size of a terminal leaf is assumed to be 20 $cm^2$, then 14,600 leaves would fit under the tree, or 500 leaves $m^{-2}$ ground. This is probably a high estimate for several reasons. First, the leaves would not fill an area that size but rather would fall overlapping one another. Secondly, unsprayed or severely scabbed trees tend to lose leaves prematurely. This would result in considerable decomposition of leaves and reduction in inoculum prior to the onset of winter. Also, other leaves from different tree and plant species could cover infected leaves later in autumn. The inoculum source in this study contained 1700 leaves, (425 leaves $m^{-2}$), and was thus the equivalent of at least one and probably two or three abandoned or unsprayed trees heavily infected with scab.

Accepting this, it can be stated that beyond 30 m of a tree heavily infected with apple scab the previous growing season, ascospores from infected leaves would be randomly dispersed and ascospore densities would be at background levels. Short of an abandoned orchard adjacent to a commercial orchard, the effects of unsprayed trees on inoculum and disease in a nearby commercial orchard would be minimized by 30 m from the inoculum source. Likewise, an individual pocket of inoculum within an orchard would not have a substantial effect in that orchard beyond 30 m. Burchill (15) detected no infection on unsprayed trees more than 15 m from an

inoculum sourcewithin the orchard, and Hirst and Stedman (54) found that airborne inoculum levels in an orchard treated with dinitro-ortho cresol to reduce overwintering inoculum were not affected by inoculum from an untreated orchard 60 m beyond the untreated orchard. With a larger inoculum source, one that might result from an untended orchard, a greater distance would be required to diminish the effects, but this distance would probably be in a range of 100 to 200 m. Keitt and Palmiter (65) examined the disease gradient from an unsprayed orchard into an adjacent orchard, and found the gradient flattened within 200 m.

The increase in disease within an orchard that results from an external inoculum source will be less than but proportional to any recorded increase in inoculum. In the present study, 86% and 91% of the lesions recorded on unprotected limbs occurred less than 10 m from the inoculum source in 1982 and 1983, respectively. Farther away, there was no difference in the severity of disease on unsprayed limbs and sprayed trees in the rest of the orchard. It was initially postulated that the low number of lesions was due to redistribution of fungicide from sprayed to unsprayed portions of trees. However, it was determined (Chapter IV) that few lesions actually result from ascospore infections, with the number of lesions on a tree much less than the number of ascospores trapped in that tree.

Several other factors also suggest that large numbers of ascospores would not be transported for long distances during an infection period. First, the air during an infection periods tends to be very heavy, with light winds. Although New England is notorious for its "Nor'easters", most spring rains usually result from passing

fronts with light winds during the rainy periods and brisk winds prior to and after (80% of infection periods from 1982 - 1985 had winds of 5 m $s^{-1}$ or less). In light winds, spores disperse more quickly vertically (46; Table 4), and diffusion of the spore cloud occurs more quickly. Secondly, the scrubbing effect of rain would increase ascospore deposition close to the source. Thirdly, most ascospores are released over a ten hour period from 0700 to 1800 hr (78) and, as shown in this study there is usually considerable variation in wind direction (Table 4) and more variation in the direction of spore dispersal (Appendix III, Fig. 1-4) over an extended period of spore release. Thus, it is unlikely that many ascospores will be released in one direction over the course of an infection period.

The rapid diffusion of airborne ascospores from a source combined with a low frequency of primary infections suggest that growers should be most concerned with inoculum originating within their orchards and not from an outside source. Because of this, growers should be able to utilize apple scab management programs with reduced fungicide applications without having to worry about outside inoculum increasing disease levels in their orchards. When untended apple trees are located near a commercial orchard, it might be more economical to adapt cultural or chemical means to reduce overwintering inoculum from those trees, or to remove the trees. This could reduce the need for early season protectant applications of fungicides that might not be needed in the absence of an inoculum source external to a commercial orchard.

# CHAPTER IV.

## QUANTIFICATION OF FOLIAR SCAB LESIONS RESULTING FROM PRIMARY APPLE SCAB INFECTIONS

### Introduction

In chapter III, research on the airborne dispersal of ascospores of V. inaequalis was presented. Spore dispersal gradients were measured and it was concluded that most inoculum in an orchard originates within the orchard. Inoculum originating outside an orchard would not contribute significantly to the amount of scab occurring within that orchard, with the possible exception of an abandoned orchard within 100-200 m.

Most apple scab management programs emphasize fungicides to control the primary phase of the disease cycle, because limiting the number of primary scab lesions will lessen the amount of secondary scab later in the growing season. Two main programs exist for scheduling fungicide applications. In a protectant program the strategy is to schedule fungicide applications based on tree growth stages, with the first application at 0.7 to 1.3 cm (1/4 to 1/2") green, and subsequent applications at tight cluster, pink, bloom, petal fall, and the first or second cover spray or until the season's supply of ascospores has been exhausted (5). Applications occur at approximately weekly intervals in a protectant program, and there are

usually 6-7 sprays during the primary season. In an eradicant program, the strategy is to schedule fungicides according to the occurrence of infection periods (97,106,122,136), utilizing fungicides that inhibit the fungus after infection has occurred.

Regardless of the strategy used, the fungicide dose in each application during the primary season usually does not vary, a practice that is seemingly based upon the belief that lesion incidence resulting from each infection period will always exceed a tolerable threshold. However, several variables determine lesion incidence: cultivar resistance to V. inaequalis, the amount of susceptible leaf tissue, the density of airborne ascospores, wind speed, rain intensity, temperature, hours of leaf wetness, and fungicide residue. The purpose of this study was to determine the relationship between airborne ascospores present during an infection period and the lesion density that results from infections by these ascospores. The effects of cluster and terminal leaf area and maturation on this relationship were also examined. In addition, scab development on the fruit was monitored throughout the growing season to determine if the occurrence of early season foliar lesions was related to the final incidence of fruit scab. The most serious economic consequences of apple scab in commercial orchards result from fruit scab, but the infection of the fruit during primary infection periods has received little attention from researchers.

## Materials and Methods

**Woodman Farm Study** In 1984 and 1985, studies were conducted in a 0.5 ha planting of semi-dwarf McIntosh apple trees (EM-7 rootstock) at the University of New Hampshire Woodman Farm. The trees were spaced at 6.1 m in rows 6.1 m apart, and were approximately 20 years old, 4-5 m tall, and pruned in a central-leader system. Each experimental plot contained 6 trees, with three trees in each of two rows (Fig. 1). One plot (D, Fig. 1) had only five trees, and a tree in Plot B died during the study in 1985.

A spray schedule (Table 1) was designed to leave a different plot unsprayed for each primary infection period. Sulfur (16.8 kg ai/ha) was applied to each plot prior to the unprotected infection period. Two characteristics of sulfur that favored its selection in this strategy were 1) its lack of after-infection activity (50) and 2) its ability to protect tissue when applied prior to an infection period combined with an ineffectiveness to prevent scab lesions for subsequent infection periods without another application (51). Based on an earlier study with sulfur (51), it was assumed that if sulfur was applied 5 or more days prior to an infection period, the increase in leaf tissue combined with rainfall and other weathering effects on sulfur during the interval from a sulfur application to a period of exposure to ascospores would result in insufficient protection during that infection period. Fungicides applied after the unprotected infection period were captan (3.36 kg ai/ha) in 1984 or maneb 4F (8.33 l/ha) in 1985. All fungicides were applied with a Solo backpack

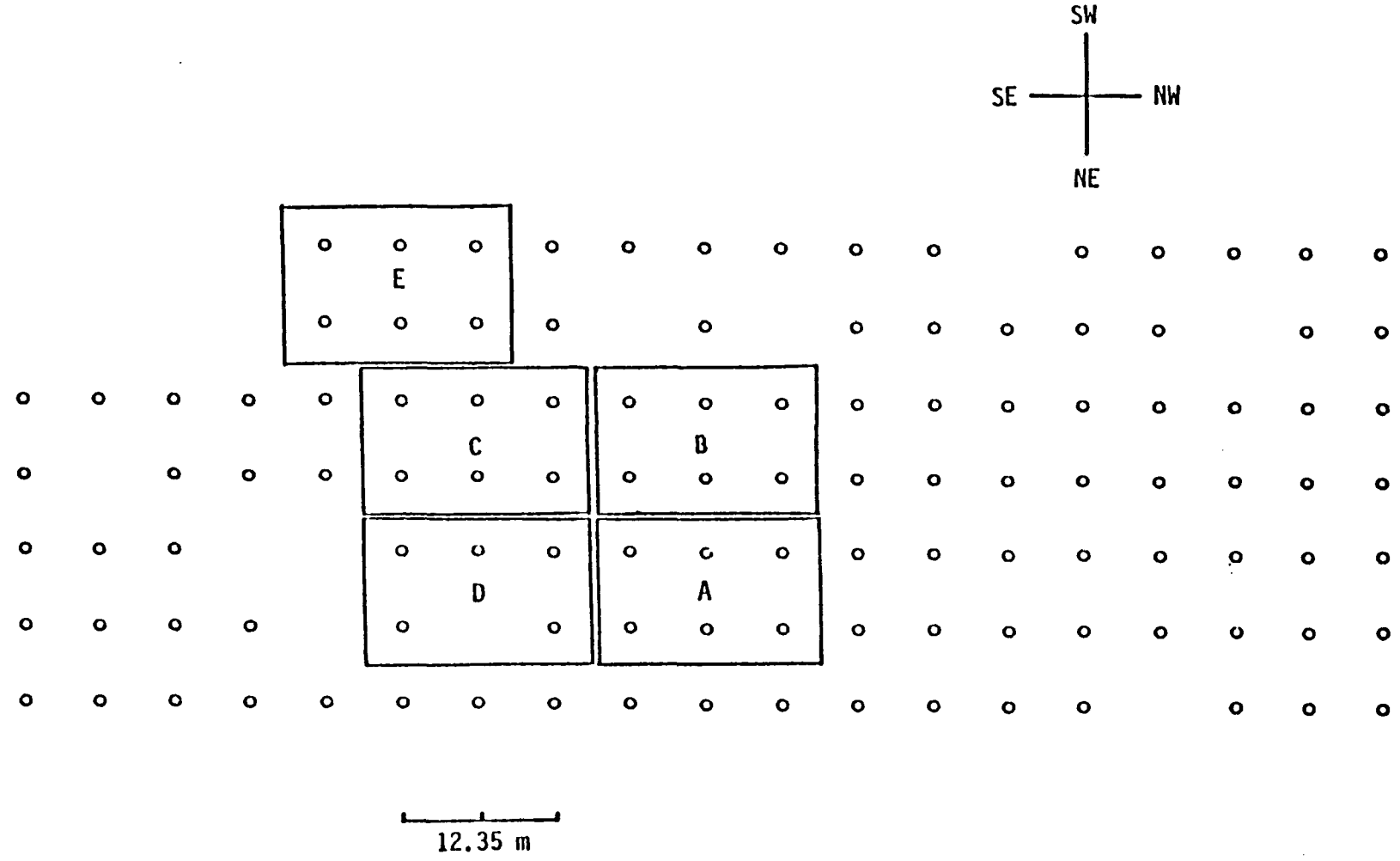



Figure 1. Plot design at Woodman Farm research orchard. Plots A-D were used in 1984; Plots A-E in 1985.

Table 1. Woodman Farm orchard fungicide spray schedule for plots left unprotected during an infection period in the primary apple scab season, 1984 and 1985.

| Plot | Fungicide applications before unprotected infection period[1] | Infection period with unprotected foliage | Fungicide applications after unprotected infection period[2] |
|---|---|---|---|
| | | 1984 | |
| A | None | 3-5 May | 7,13,18,25 May; 4 June |
| B | 29 April | 8-9 May | 13,18,25 May; 4 June |
| C | 29 April; 7 May | 12-14 May | 18,25 May; 4 June |
| D | 29 April; 7,13, May | 28 May - 2 June | 4 June |
| | | 1985 | |
| A | None | 5-7 May | 9,21,30 May; 4,11 June |
| B | 23 April; 1 May | 18-19 May | 21,30 May; 4,11 June |
| C | 23 April; 1,15[3] May | 21-22 May | 24,30 May; 4,11 June |
| D | 23 April; 1,9,21 May | 27-29 May | 30 May; 4,11 June |
| E | 7,15,24 May | 5-7 June | 11 June |

[1]Sulfur at 16.6 kg ai/ha applied to plots prior to the infection period when foliage was unprotected.

[2]Captan at 3.36 kg ai/ha (1984) of maneb 4f at 8.33 l/ha (1985) was applied to each plot for the remainder of the primary season.

[3]Triforine at 2.21 l ai/ha was applied because it has a period of protection of only 3-5 days (105) and weather forecasts indicated that several storm fronts would be passing through the area from 16 to 23 May.

sprayer (D-7032 Sindelfingen 6, Box 20, W. Germany) during the primary season.

Six "Zuck" volumetric spore traps designed by Zuck (135,136, Appendix A) were placed at each of four trees within the designated plot to trap airborne ascospores during each infection period (Appendix E, Fig. 1). At each tree, three traps were placed 0.9 m above ground level under the drip line and three traps were placed within the canopy 3 m above ground level. The spore traps were powered by 12 VDC motors (Herbach and Rademan Inc, Philadelphia, PA) run by DC power (Appendix B). Intervals of trap operation were controlled by a leaf wetness indicator (Appendix B) and recorded on a modified hygrothermograph by a pen arm that responded to the activity of the leaf wetness indicator (81).

The trapping surface for each trap was a 25 x 75 mm glass slide. The slide was not coated with an adhesive, because ascospores of V. inaequalis are adherent (64). Slides in the lower (0.9 m) spore traps were changed daily at 0700, 1200, and 1800 hours, Eastern Standard Time (EST). If there was a heavy downpour at the designated time, the slides were changed after the rain intensity diminished. Slides in the higher (3 m) spore traps were not changed until the conclusion of an infection period because of the time involved and for safety purposes. Spores were deposited on each slide in a thin band corresponding to the orifice of the spore trap (Appendix A, Fig. 2). The spore deposits were sealed in a solution of Gelvatol:water:glycerol (35g:100ml:50ml) for storage. The entire band on each slide was scanned and the ascospores deposited were recorded as 'ascospores per slide'. Airborne ascospore density (ascospores $m^{-3}$

of air) was calculated by dividing the ascospores per slide by the cubic meters of air drawn through the trap for the sampling period.

The inoculum source contained approximately 1700 leaves heavily infected with V. inaequalis the previous year. The leaves were removed from unsprayed trees at the Mast Road research orchard and overwintered in wire cages placed on the orchard floor. The cages were transported to the Woodman Farm prior to green tip in each growing season and arranged in a circle approximately 2.8 m in diameter in the center of the plot selected for the first infection period (Appendix E, Fig. 1). A "Zuck" trap and a recording volumetric spore trap (36) were placed within the circle. Following the first infection period the spore traps and the cages with inoculum were placed in the plot left unprotected for the second infection period. This procedure was repeated for each primary infection period. To minimize background inoculum, the entire orchard had been treated with a post- harvest application of benomyl (900 g ai/ha) just prior to leaf fall in 1983 and 1984 to reduce overwintering inoculum. Benomyl reduces the overwintering inoculum by >99% (37).

**Leaf area measurement.** Leaf area was estimated by sampling 30 terminals and 30 clusters each week. The leaves from each terminal and cluster were removed and measured with a Licor portable leaf area meter (Model LI-3000, Licor Inc, Lincoln, NE 68504). Regression equations relating terminal and cluster growth with time were used to estimate the leaf area for infection periods that occurred between sampling dates. Leaf area measurements and calculations were for one leaf surface only. It was assumed that the upper and lower leaf

surfaces were susceptible, so the calculated leaf area was doubled to estimate the amount of susceptible terminal or cluster leaf tissue when computing lesions $cm^{-2}$.

**Monitoring environmental variables.** Temperature ($^{o}F$), relative humidity (%), rainfall (hundreths in.), and hours of leaf wetness were recorded continuously at the Woodman Farm using weather monitoring instrumentation developed by MacHardy and Sondej (81). Mean temperature during wet periods was determined using the technique developed by MacHardy (77). Wind speed and direction were recorded using a recording anamometer (Weathermeasure Corp., Sacremento, CA.) Rain intensity was assessed in accordance with the International Code of Weather Symbols (91).

**Foliar and fruit scab assessment.** Fifty terminals and 50 clusters on each tree in each plot were assessed for foliar scab: 25 (designated upper terminals/upper clusters) were >1.8 m above the ground, and 25 (designated lower terminals/lower clusters) were <1.8 m above the ground. The number of lesions on each infected leaf was recorded for each terminal and cluster. Cluster and terminal lesion densities were calculated by dividing the total number of lesions recorded on each tree by the leaf area (for the sample size) present during the infection period for which the trees were unprotected. If the mean number of leaves per terminal during an infection period was greater than seven, a second lesion density was calculated using 12 $cm^2$, the mean area of the youngest seven leaves. In seedling studies described in this chapter and in other studies (104,121), lesion development was

restricted to the youngest seven leaves, so it was assumed that this would also be true in the present study. Utilizing a calculation that considered only the leaf tissue that was sufficiently susceptible to provide a measurable lesion density with the low level of inoculum present provided a more accurate measure of the relationship between airborne ascospores and lesion density. Fruit scab was assessed by examining one hundred fruit per tree and recording the percentage of fruit with scab.

**Seedling Study.** In 1984 and 1985, McIntosh apple seedlings were placed in the Mast Road research orchard during primary infection periods to establish a relationship between airborne ascospore density and lesion density. A Burkhard 7-day recording volumetric spore trap was used to monitor airborne ascospore densities. Apple seedlings were grown from seed gathered from open pollinated McIntosh apples harvested from a McIntosh-Cortland orchard. The seeds were germinated in perlite and left on a propagation bench for 3-4 weeks before transplanting to 10.2 cm standard pots filled with a mixture of sterilized, recycled soil and vermiculite, 4:1. All seedlings had 8-10 fully expanded leaves when placed in the orchard. Twelve seedlings were placed in the orchard approximately 1 m from the Burkhard spore trap from 0700 - 1200 hours, 1200-1700 hours, or 1700-0700 hours during infection periods. The seedlings were then placed in a mist chamber at 15 C for at least 12 hours, conditions that allowed ascospores on the leaf surface to infect the leaf tissue.

Leaf area of the seedlings was calculated using a leaf area index developed as follows. One hundred and twenty leaves were removed from

seedlings at the junction of the petiole and the base of the leaf. The length (L) and width (W) of each leaf was measured with a metric rule, and the leaf area (A) was measured with a Licor portable leaf area meter. A leaf area index (I) was calculated for each leaf using the equation I = A/LW. The mean leaf area index for all leaves was 0.71 (s.d. = 0.0885). The mean difference between the estimated leaf area and the measured leaf area for all leaves was 0.69 $cm^2$, with the percent error decreasing as the size of the leaf increased (Fig. 2). When seedlings were removed from the mist chamber, the lengths and widths of the top 7 leaves were measured and the area of each leaf calculated using the equation A = 0.71LW. The youngest unfurled leaf with measurable length and width was marked with string for easy identification, and the plants were incubated for 20 days in the greenhouse. Szkolnik's numbering system (121) was used to identify the position of each leaf when the seedlings were indexed for scab lesions at the end of the incubation period (Fig. 3).

**1985 Mast Road fungicide trials.** A study to examine the effect of a delayed starting date of the fungicde spray program on the incidence of foliar and fruit scab was conducted at the Mast Road research orchard in 1985. Several strategies for scheduling fungicide sprays were employed (Table 2) on mature, fruit-bearing trees: three McIntosh and three Cortland per schedule. One strategy (schedule A, Table 2) followed a standard protectant program, applying a fungicide at 7-10 day intervals throughout the primary season, beginning at 0.6-1.3 cm green. A second strategy (schedules B and C, Table 2) applied the first fungicide after one, two or three infection periods had occurred

(Table 2, schedules B and C, D and E, and F, respectively). Schedules B and D scheduled different materials than schedules C and E for the first two fungicide treatments, with maneb 4F (8.33 l $ha^{-1}$) or dodine (0.81 kg ai $ha^{-1}$) applied for schedules B and D or C and E, respectively. All trees in sprayed plots were treated at approximately two-week intervals after the conclusion of the primary scab season. Check trees received no fungicide throughout the growing season.

Similar strategies were employed on young, non-bearing McIntosh trees to study only foliar scab build-up. In one strategy, trees were left unsprayed for the first, first two, first three, or first four infection periods, (Table 2, schedules G-J, respectively). In a second strategy, no fungicide was applied after the first, second, third, fourth or fifth infection periods (Table 2, schedules K-M, respectively).

On bearing and non-bearing trees, 25 terminals and 25 clusters on each tree were assessed for foliar scab. Fifty fruit were assessed for scab on each bearing tree.

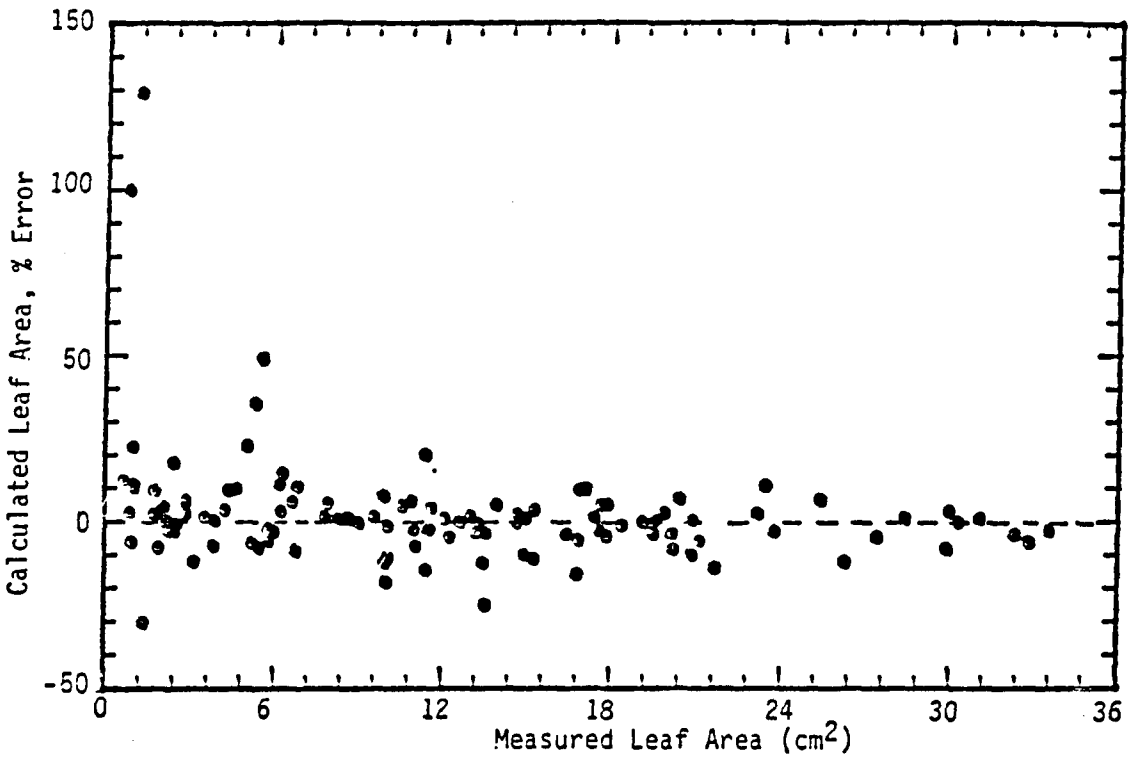



Figure 2. Accuracy of a leaf area index in estimating leaf area of McIntosh seedlings. The measured leaf area was determined using the equation A = L x W x I, where L = leaf length, W = leaf width, and I = leaf area index = 0.71.

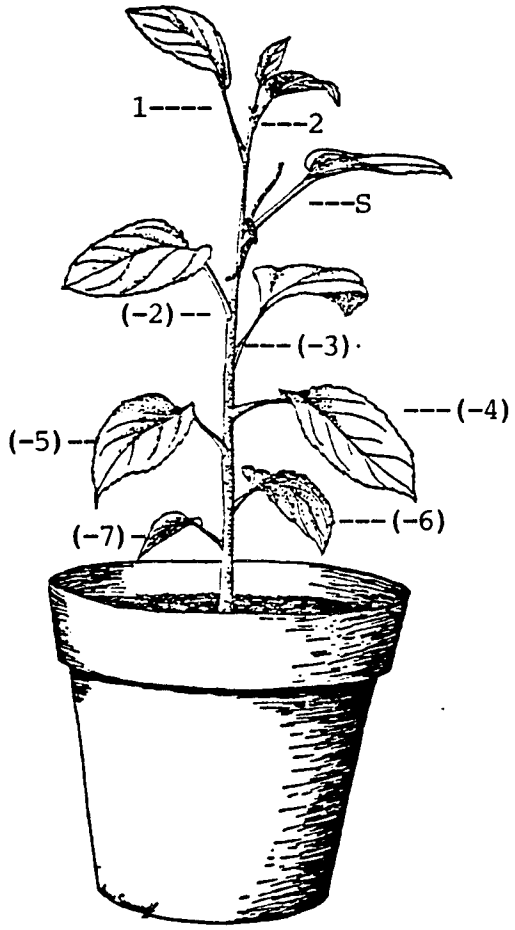



Figure 3. Leaf position designations on McIntosh seedlings after an incubation period of 20 days. The string leaf (S) was the youngest, unfurled leaf when the seedlings were placed in the orchard. When the seedlings were assessed for scab, leaves (S) - (-3) included any leaves above the string leaf which developed lesions, the string leaf, and the two leaves below the string leaf. Leaves (-4) - (-7) included the third, fourth, fifth and sixth leaves below the string leaf.

Table 2. Spray schedules for 1985 Mast Road orchard studies.

| Schedule | Strategy during primary scab season | Spray dates |
|---|---|---|
| | Bearing trees | |
| A | Protectant schedule, spray at 7-10 day intervals. | 23 April; 1,9,15,22,30 May; 4,11,29 June; 16 July; 2 Aug. |
| B + C | Leave unprotected for first infection period. B = maneb, first 2 sprays; C = dodine, first 2 sprays. | 9,15,22,30 May; 4,11,29 June; 16 July; 2 Aug. |
| D + E | Leave unprotected for first two infection periods. D = maneb, first 2 sprays; E = dodine, first 2 sprays. | 21,30 May; 4,11,29 June; 16 July; 2 Aug. |
| F | Leave unprotected for first three infection periods. | 24,30 May; 4,11,29 June; 16 July; 2 Aug. |
| P | Unsprayed check. | |
| | Non-bearing trees | |
| G | Leave unprotected for first infection period. | 9,15,22,30 May; 4,11,29 June; 16 July; 2 Aug. |
| H | Leave unprotected for first two infection periods. | 21,30 May;4,11,29 June; 16 July; 2 Aug. |
| I | Leave unprotected for first three infection periods. | 24,30 May; 4,11,29 June; 16 July; 2 Aug. |
| J | Leave unprotected for first four infection periods. | 30 May; 4,11,29 June; 16 July; 2 Aug. |
| K | No protection after the first infection period. | 23 April; 1 May |
| L | No protection after the second infection period. | 23 April; 1,9,15 May |
| M | No protection after the third infection period. | 23 April; 1,9,15,22 May |
| N | No protection after the fourth infection period. | 23 April; 1,9,15,22 May |
| O | No protection after the fifth infection period. | 23 April; 1,9,15,22,30 May; 4 June. |

## Results

The 1984 and 1985 primary apple scab seasons were studies in meteorological contrast. The 1984 primary season was extremely wet, climaxed by a 6-day primary infection period when 17.0 cm of rain fell (Table 3) followed by a dry summer with 11 secondary infection periods from 4 June through 21 August. In contrast, the 1985 primary scab season was very dry. From 1 May through 6 June only 7.1 cm of rain fell, and there was an 11-day dry period from 7-18 May when tree phenology advanced from pink through petal fall (Table 3). The summer was wetter than 1984 with 22 secondary infection periods between 7 June and 31 August.

Tree growth stages occurred approximately one week earlier in 1985 than in 1984, but due to drier weather in 1985, 95% of the season's ascospores were trapped after petal fall (Fig. 4). In 1984 all ascospores were trapped by the completion of petal fall. In both years, cluster leaves were fully expanded just prior to or shortly after bloom (Fig. 4). Terminal leaves were absent or expanding slowly until pink (Fig. 4), but between pink and the end of the primary season the terminal leaf area increased 400 to 600%.

Table 3. Summary of tree floral phenology and meteorological conditions at the Woodman Farm orchard during rainy intervals in the primary apple scab season, 1984 and 1985.

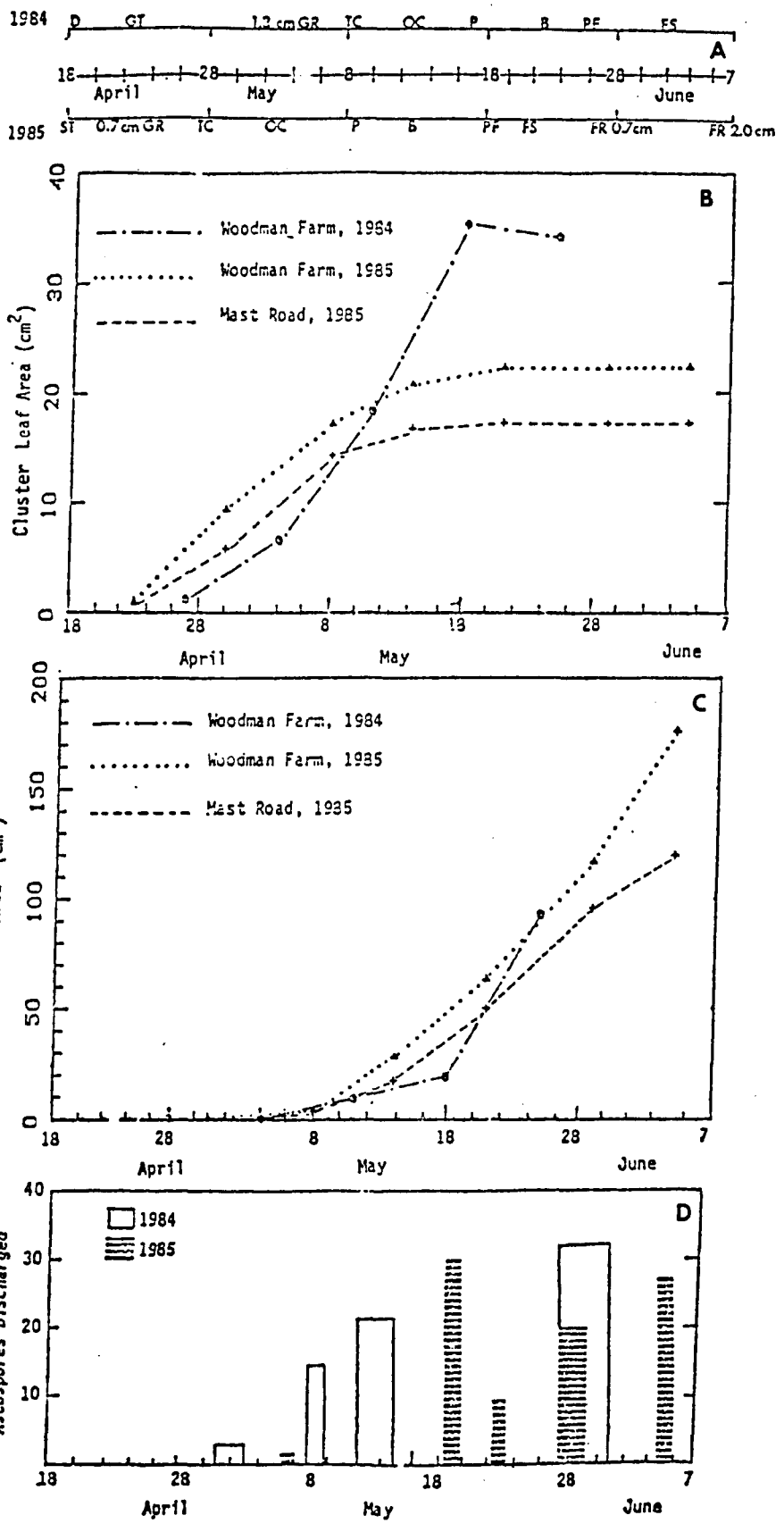
[1]Growth stages: D = dormant; GT = green tip; GR = green; TC = tight cluster; OC = open cluster; EP = early pink; P = pink; B = bloom; PF = petal fall; FS = fruit set; FR = fruit.

[2]Night rains, no ascospores released.

[3]Two periods of leaf wetness separated by a 4.5 h dry period.

| Date | Tree phenology: Floral growth stage[1] | Leaf area ($cm^2$): Cluster | Leaf area ($cm^2$): Terminal | Meteorological conditions: Rain (cm) | Leaf wetness (h) | Average Temp (C) |
|---|---|---|---|---|---|---|
| April 1984 | | | | | | |
| 18-20 | D | 0.0 | 0.0 | 1.21 | 45 | 7.7 |
| 23-25 | GT | 0.0 | 0.0 | 3.45 | 49 | 7.2 |
| May | | | | | | |
| 3-5 | 1.3 cm GR | 8.3 | 0.0 | 4.34 | 41 | 4.5 |
| 8-9 | TC | 12.6 | 5.3 | 2.21 | 19 | 7.0 |
| 12-13 | OC | 21.3 | 9.3 | 0.68 | 21 | 11.5 |
| 13-14 | OC | 21.3 | 9.3 | 1.14 | 17 | 8.0 |
| 15 | OC | 22.1 | 11.2 | 0.07 | 8 | 9.0 |
| 16 | OC/P | 24.3 | 13.5 | 0.07 | 4 | 7.0 |
| 17 | P | 24.8 | 16.3 | <0.01 | 1 | 9.0 |
| 23[2] | B | 34.2 | 49.7 | 0.96 | 9 | 15.5 |
| 26[2] | PF | 34.2 | 87.4 | 0.96 | 7 | 13.0 |
| 28-2 June | PF-FS | 34.2 | 126.4 | 17.0 | 140 | 13.0 |
| April 1985 | | | | | | |
| 22 | 0.7 cm GR | 1.0 | 0.0 | 0.68 | 4 | 10.0 |
| 28 | TC | 7.2 | 0.0 | 0.02 | 5 | 7.2 |
| May | | | | | | |
| 3-4 | OC | 11.1 | 1.0 | 1.57 | 28.5 | 3.9 |
| 5-7 | EP | 12.6 | 1.2 | 1.44 | 39.5 | 6.1 |
| 18-19 | PF | 22.3 | 69.0 | 1.75 | 35.0 | 7.7 |
| 21-22 | FS | 22.3 | 86.0 | 1.27 | 16.5 | 13.9 |
| 27-29 | FR 0.7 cm | 22.3 | 120.0 | 0.81 | 38.5 | 11.7 |
| June | | | | | | |
| 1 | FR 1.3 cm | 22.3 | 148.0 | 0.38 | 7.0 | 15.0 |
| 3 | FR 1.3 cm | 22.3 | 159.4 | <0.01 | 0.0 | 14.4 |
| 5-7 | FR 1.3-1.9 cm | 22.3 | 170.7 | 0.61 | 21 + 14[3] | 10.5 |

Figure 4. Increases in the leaf area of McIntosh clusters and terminals and their relationship to tree floral phenology and ascospore discharge during the primary season, 1984 and 1985. A: Tree floral phenology; B: Increase in cluster leaf area; C: Increase in terminal leaf area; D: Percentage of season's supply of ascospores released during each infection period. Growth stages: D = dormant; ST = silver tip; GR = green tip; GR = green; TC = tight cluster; OC = open cluster; P = pink; B = bloom; PF = petal fall; FS = fruit set; FR = fruit.


1984
D
GT
1.2 cm GR
TC
CC
P
B
PF
FS
A
18
28
8
18
28
7
April
May
June
1985
ST
0.7 cm GR
TC
CC
P
B
PF
FS
FR 0.7 cm
FR 2.0 cm
B
Woodman Farm, 1984
Woodman Farm, 1985
Mast Road, 1985
Cluster Leaf Area (cm²)
0
10
20
30
40
C
Woodman Farm, 1984
Woodman Farm, 1985
Mast Road, 1985
Terminal Leaf Area (cm²)
0
50
100
150
200
D
1984
1985
% Season's Ascospores Discharged
10
20
30
40

## Woodman Farm Orchard Study

A meteorological summary of each primary infection period is presented in Appendix D. The number of ascospores trapped and the number of lesions recorded for each infection period are presented in Appendix E.

**Cluster lesion density.** The number of lesions recorded for 250 or 300 clusters in each plot was low: 6, 10, 18, and 5 lesions, respectively in plots A, B, C, and D in 1984 and 8 and 5 lesions recorded in plots A and E in 1985 (Table 4). Cluster lesion densities for trees unprotected for different primary infection periods in 1984 and 1985 are presented in Table 5. Lesion density ranged from 0 to $1.4 \times 10^{-3}$ lesions $cm^{-2}$ of cluster leaf tissue.

Although the differences in cluster lesion densities were not statistically significant, the greatest densities resulted from infection periods that occurred prior to bloom when the cluster leaves were expanding. In 1985, no cluster lesions were recorded for 3 plots unprotected after petal fall (B, C and D, Table 4). Plot E (Table 4) had cluster lesions, but trees in this plot were unprotected during the first (5-7 May) and fifth (5-7 June) infection periods. Although the date of the cluster leaf infections in plot E cannot be definitely ascertained, in all probability those infections occurred during the first infection period (see discussion).

**Terminal lesion density.** In 1984, terminal lesions were recorded on 1 July for trees in each plot (Appendix E, Fig. 2a-d). The number of

Table 4. Cluster and terminal foliar scab incidence and severity, and fruit scab (%) on trees unprotected by fungicide for one primary infection period in 1984 and 1985.

[1]Date of infection period when trees were unprotected.

[2]GR = green; TC = tight cluster; OC = open cluster; PF = petal fall; FS = fruit set; FR = fruit.

[3]Percentage of cluster or terminal leaves with scab.

[4]Number of lesions recorded for 50 clusters or terminals per tree, 5 or 6 trees per plot.

Means followed by the same letter do not differ significantly according to Student-Newman-Keuls Test.

ns = not significant. *** Significant at p = 0.01. * Significant at p = 0.1.

| Plot | Date[1] | Tree floral phenology[2] | Cluster scab | | Terminal scab | | | | % Scabbed fruit | | # Scabbed fruit | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Incidence[3] | Severity[4] | Incidence | | Severity | | | | | |
| | | | | | 1 July | 1 Sept | 1 July | 1 Sept | 1 July | 1 Sept | 1 July | 1 Sept |
| | | | | | 1984 | | | | | | | |
| A | 3-5 May | 1.3 cm GR | 1.3 ns | 6 | 0.3 a*** | | 52 | | 1.0 ns | 1.0 ns | 8 | 6 |
| B | 8-9 May | TC | 2.7 | 10 | 0.3 a | | 32 | | 1.3 | 0.7 | 7 | 1 |
| C | 12-14 May | OC | 3.6 | 18 | 0.9 a | | 99 | | 1.2 | 0.2 | 1 | 1 |
| D | 28 May - 3 June | PF-FS | 2.0 | 5 | 2.5 b | | 356 | | 1.6 | 2.6 | 8 | 13 |
| | | | | | 1985 | | | | | | | |
| A | 5-7 May | 0.6 cm GR | 2.6 b* | 8 | 0.0 a*** | 0.5 ns | 0 | 38 | 0.0 ns | 0.1 ns | 0 | 7 |
| B | 18-19 May | PF | 0.0 a | 0 | 0.0 a | 0.2 | 0 | 20 | 0.0 | 0.0 | 0 | 0 |
| C | 21-22 May | FS | 0.0 a | 0 | 0.1 a | 0.1 | 5 | 4 | 0.0 | 0.0 | 0 | 0 |
| D | 27-29 May | FR 0.6 cm | 0.0 a | 0 | 0.1 a | 0.6 | 4 | 27 | 0.0 | 0.0 | 0 | 0 |
| E | 5-7 June | FR 1.2-1.8 cm | 1.3 b | 5 | 0.5 b | 0.6 | 29 | 65 | 0.6 | 0.5 | 4 | 3 |

Table 5. The relationship between area dose and lesion density in trees unprotected with fungicide during primary infection periods in 1984 and 1985.

[1]Date of infection period when trees were unprotected.

[2]GR = green; TC = tight cluster; OC = open cluster; PF = petal fall; FS = fruit set; FR = fruit.

[3]Leaf area present during the infection period.

[4]Lesions $cm^{-2}$ x $10^{-3}$ of susceptible leaf tissue.

[5]Ascospores $cm^{-2}$

[6]Cluster lesion density based on cluster leaf area during first infection period.

***Significant at p = 0.01. **Significant at p = 0.05. *Significant at p = 0.1. ns = not significant.

| Plot | Date[1] | Tree floral phenology[2] | Leaf area ($cm^2$)[3] | | Lesion density[4] | | | Area[5] Dose | Ascospores per lesion | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | Cluster | Terminal | Cluster | Terminal | | | Cluster | Terminal |
| | | | | | | 1 July | 1 Sept | | | |
| | | | | | 1984 | | | | | |
| A | 3-5 May | 1.3 cm GR | 8.3 | 12.6 | 1.2 ns | 0.0 a*** | | 1.54 | 1,283 | — |
| B | 8-9 May | TC | 12.6 | 5.3 | 1.2 | 9.8 a | | 0.51 | 425 | 52.0 |
| C | 12-14 May | OC | 21.3 | 9.3 | 1.4 | 26.3 b | | 0.47 | 336 | 17.8 |
| D | 28 May - 3 June | PF-FS | 34.1 | 126.4 | 0.4 | 59.3 b | | 0.42 | 10,500 | 7.1 |
| | | | | | 1985 | | | | | |
| A | 5-7 May | 0.6 cm GR | 12.6 | 1.2 | 1.1 c** | 0.0 a*** | 0.3 ab* | 0.82 | 745 | — |
| B | 18-19 May | PF | 22.3 | 69.0 | 0.0 a | 0.0 a | 0.1 ab | 0.49 | — | — |
| C | 21-22 May | FS | 22.3 | 86.0 | 0.0 a | 0.7 b | 0.02 a | 0.001 | — | 1.4 |
| D | 27-29 May | FR 0.6 cm | 22.3 | 120.0 | 0.0 a | 0.7 b | 0.2 ab | 0.13 | — | 185.0 |
| E[6] | 5-7 June | FR 1.2-1.8 cm | 22.3 | 170.7 | 0.7 b | 4.0 b | 0.8 b | 0.02 | — | 5.0 |

lesions recorded in plots A-D were 52, 32, 99, and 356, respectively (Table 4). In plot A, lesions were recorded on terminal leaves although no terminal leaves were present during the infection period on 3-5 May when these trees were unprotected. Terminal lesions in plot B were recorded for leaves 4-10, leaf 1 being the oldest, although leaves 7-10 were not present when the trees were unprotected (8-9 May). Half the lesions in plot C occurred on two leaves in the lower canopy of one tree (Appendix E, Fig 2c) which was upwind of the inoculum source during the 8-9 May infection period. Some leaves in plots A, C and D contained sheet scab (Appendix E, Fig 1a,c,d) which is usually indicative of infections by conidia.

There were 97% fewer terminal lesions recorded in 1985 than in 1984. On 1 July, 1985, no terminal lesions were recorded for plot A or B, and 5, 4, and 29 terminal lesions were recorded for plots C, D and E, respectively (Table 4). No leaves had sheet scab, and all recorded lesions were on leaves that were present during infection periods when their respective trees were unprotected with fungicide.

Terminal lesion densities for each plot in 1984 and 1985 are presented in Table 5. Densities were much higher in 1984, reflecting more lesions recorded and less leaf area present during 1984 primary infection periods. When the terminal leaf area was adjusted to include only the youngest seven leaves, the lesion density was still 2-15 times greater in 1984 than the highest density (plot E) recorded in 1985. In both years, terminal lesion density based on the area of susceptible tissue during an infection period was statistically significantly lower for infection periods occurring at the beginning of the primary season than those at the end of the primary season.

**Secondary Scab Build-up, 1985.** Scab was reassessed on 1 Sept to determine if the percentage of terminal leaves with scab lesions and the density of terminal lesions had changed since 1 July. When calculating terminal lesion density, a value of 424 $cm^2$, which Gadoury (38) had determined was the mean area of McIntosh terminal leaves at harvest, was used. This value was approximately 3.5 times the leaf area at the end of the primary season. Thus, a lesion density of 0.22 x $10^{-3}$ lesions $cm^{-2}$ on 1 Sept represented 3.5 times as many lesions as an equivalent density on 1 July. On 1 Sept, the greatest lesion density (0.8 x $10^{-3}$) occurred in plot E and the smallest (0.02 x $10^{-3}$) in plot C (Table 5). An increase in terminal lesion density was recorded only in plots A and B, but neither plot had terminal leaf scab on 1 July. The percentage of scabbed leaves increased in all plots except plot C (Table 4), but the percentage of terminals with one or more lesions was not correlated with the total number of lesions per plot (Table 4). In plot E, the number of lesions increased nearly 3-fold, but the percentage of scabbed leaves increased only slightly, from 0.5% on 1 July to 0.6% on 1 Sept. Plot D had the same percentage of scabbed leaves as plot E, but 1/3 the number of lesions.

**Fruit scab assessment.** Scab was observed on fruit in each plot in 1984, with 8, 7, 1 and 8 scabbed fruit per 600, 600, 540, and 500 fruit examined on 15 June in plots A-D, respectively (Table 4). When fruit was reexamined on 2 Aug., the number of scabbed fruit in plots A-D was 6, 1, 1, and 13, respectively. Fewer scabbed fruit were observed in 1985, with 4 scabbed fruit (600 examined) recorded for

plot E and none in plots A-D on 1 July. There was still no fruit scab observed on trees in plots B, C, and D on 1 Sept., but 7 scabbed fruit (1.2%) were recorded in plot A and 3 scabbed fruit (0.5%) in plot E (Table 4).

**Airborne ascospores.** The number and density of ascospores trapped at the inoculum source and the average number and density trapped in tree canopies approximately 2-6 m from the source are presented in Table 6. The number of ascospores trapped by each trap during each infection period is presented in Appendix E, Figure 2.

The total number of ascospores trapped at the source for each infection period ranged from 130 to 154 ascospores in 1984 (Table 6). Airborne ascospore density at the source, which reflects the time over which spores were trapped as well as the number of spores trapped, ranged from 4.4 to 16.3 ascospores $m^{-3}$ air. In 1985, there was much greater variation in the number of ascospores trapped and the airborne ascospore density at the source during each infection period. The number of ascospores trapped at the source ranged from 1 to 157, and the airborne ascospore density ranged from 0.2 to 11.5 ascospores $m^{-3}$ air.

The average number and density of ascospores trapped 2-6 m from the inoculum source was, with one exception on 5-7 May, 1985, much less than those trapped within the source. The greatest average airborne ascospore density in the canopy in 1984 and 1985 was 0.08 and 0.09 ascospores $m^{-3}$ air, respectively. The mean ratio of airborne ascospore density recorded at the source to that recorded in the canopy was 223:1 and 107:1 in 1984 and 1985, respectively. Excluding

Table 6. Airborne ascospore density and ascospore area dose at a point source of ascospores and within the canopies of 4 trees not more than 6 m from the point source.

[1]Date of infection period when spores were trapped.

[2]Total ascospores collected by a "Zuck" volumetric spore trap 0.5 m above a point source of inoculum containing approximately 1700 heavily scabbed leaves (Source) and the average number of ascospores trapped by 24 "Zuck" traps placed within tree canopies (Canopy).

[3]Ascospores $m^{-3}$ air trapped during each infection period.

[4]Ascospores $cm^{-2}$, calculated using the equation Area dose = Spores $m^{-3}$ x wind speed (m $s^{-1}$) x sampling period (s).

| Plot | Date[1] | Sampling period (h) | Mean wind speed (m sec-1) | Ascospores trapped[2] | | Airborne ascospore density[3] | | Ascospore area dose[4] | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | Source | Canopy | Source | Canopy | Source | Canopy |
| | | | | 1984 | | | | | |
| A | 3-5 May | 10 | 5.7 | 154 | 8.0 | 13.5 | 0.08 | 277.0 | 1.54 |
| B | 8-9 May | 7 | 3.8 | 130 | 5.1 | 16.3 | 0.05 | 379.1 | 0.51 |
| C | 12-14 May | 17 | 2.0 | 138 | 8.1 | 7.1 | 0.04 | 86.9 | 0.47 |
| D | 28 May - 2 June | 26.5 | 2.3 | 132 | 3.7 | 4.4 | 0.02 | 96.5 | 0.42 |
| | | | | 1985 | | | | | |
| A | 5-7 May | 12 | 5.0 | 5 | 5.8 | 0.4 | 0.04 | 8.6 | 0.82 |
| B | 18-19 May | 10 | 2.0 | 77 | 8.3 | 6.8 | 0.07 | 48.6 | 0.49 |
| C | 21-22 May | 4 | 0.5 | 1 | 0.1 | 0.2 | 0.001 | 0.1 | 0.001 |
| D | 27-29 May | 12 | 1.0 | 157 | 13.7 | 11.5 | 0.09 | 49.7 | 0.13 |
| E | 5-7 June | 14 | 1.4 | 32 | 3.4 | 2.0 | 0.02 | 14.1 | 0.02 |

the infection period on 21-22 May, 1985, the densities of airborne ascospores recorded in the canopy were similar both years, ranging from 0.02 to 0.09 ascospores $m^{-3}$ air.

**Relating airborne ascospore density to lesion density.** Within individual plots there was no consistent relationship between the number of ascospores trapped in a tree and the lesions recorded in that tree (Appendix E). For example, on 8-9 May, 1985, the number of ascospores trapped in each of two trees was 34 and 36 (Appendix E, Fig. 2b), while the number of cluster lesions recorded for each tree was one and four, respectively. On 27-29 May, 1985, (Appendix E, Fig. 2h) two trees in which 9 and 115 ascospores were trapped each had one terminal lesion.

In 1984 the ratio of airborne ascospores to cluster lesions decreased with each successive infection period until the final infection period on 27 May - 2 June when the largest ratio was recorded (Table 5). In 1985, cluster lesions were only recorded in two plots (A and E) unprotected for the infection period prior to bloom on 5-7 May. The ratio of 745 ascospores for each cluster lesion in plot A was within the range of 336 to 1,283 ascospores per cluster lesion recorded for the first 3 infection periods in 1984. There were no traps in plot E during that period, so a ratio could not be calculated.

The ratio of airborne ascospores to terminal lesions in 1984 also decreased with each successive infection period, but the ratios were much lower than the ratios on cluster leaves that year. It was difficult to detect any trends in the ascospore/lesion ratio in 1985

because no terminal lesions were recorded for the first two infection periods (plots A and B), and in plot C, which was unprotected for the third infection period, only 2 ascospores were trapped by the 24 spore traps located in the tree canopies which resulted in a low ratio of 1.4 ascospores per terminal lesion.

**Mast Road study, 1985.** Because of an 11-day dry interval from 7-18 May, during which the trees continued to develop, trees not scheduled to be sprayed until after the second or third primary infection period were unprotected with fungicide until after bloom. Trees sprayed at the pink bud phase were unprotected for one infection period (5-7 May), and those on a protectant schedule were protected for all infection periods after bud break.

Cluster lesions were recorded for all McIntosh and Cortland spray schedules (Table 7). The lowest scab lesion incidence and density were recorded for trees sprayed according to the protectant schedule (schedule A). There were no significant differences ($p = 0.1$) in cluster leaf scab on trees which had been unprotected for at least the first infection period, even though trees unprotected for two (schedules D and E), three (schedule F), or five (unsprayed) infection periods had been exposed to 37%, 54%, and 202% more ascospores, respectively. The cluster leaves had stopped expanding between the infection periods on 5-7 May and 18-19 May.

On 27 June, the incidence and density of scab lesions on McIntosh terminal leaves were significantly higher on unsprayed trees than on trees which had received at least one fungicide spray (Table 8). The incidence of terminal scab was greater on trees which were unprotected

for two infection periods (schedules D and E) than on trees unprotected for one infection period (schedules B and C), but less terminal scab was recorded on terminals unprotected for three infection periods (F) versus two. By 27 Aug, scab incidence and severity increased on trees in every schedule, with a significantly higher incidence and density on trees which were not sprayed with fungicide until after bloom (Schedules D, E, and F).

The percentage of scabbed fruit on 1 July ranged from 0 to 7% on McIntosh and 0 to 5% on Cortland trees (Table 7). There were no significant differences in the percentage of scabbed fruit between trees which had been unprotected for one, two, three, or five infection periods on either cultivar. No fruit scab was observed on trees in the protectant schedule.

On 24 Aug, the percentage of scabbed fruit on McIntosh had increased in some trees (schedules A, D, E, P) and decreased in others (schedule B, C, F). Scabbed fruit on unsprayed McIntosh trees increased from 1.6 to 41% between 1 July and 24 Aug. On treated trees, the greatest increase in the percentage of scabbed fruit between 1 July and 24 Aug occurred in trees that were not sprayed until fruit set on 21 May (D and E, Table 8). The percentage of scabbed fruit on Cortland increased in most trees first sprayed at pink (schedules C, D, E, F) or left unsprayed (schedule P) but did not change in trees first sprayed at 1.3 cm green (schedule A) and some trees first sprayed at pink (schedule B). On treated trees, the scabbed fruit ranged from 0 to 4.3%, with trees unprotected for three infection periods (schedule F) having significantly more fruit scab than trees unprotected for none or one infection period

Table 7. The effect of spray program starting date on cluster and fruit scab lesion development on McIntosh and Cortland trees at the Mast Road research orchard, 1985.

[1]From Table 2.

[2]Number of unprotected infection periods prior to the first fungicide application.

[3]Noted for the last infection period prior to the first spray. ST = silver tip; GR = green; FS = fruit set.

[4]Cummulative ascospores $m^{-3}$ air trapped prior to the first spray.

[5]Percentage of clusters with scab.

[6]Number of lesions per 75 clusters (50 clusters for unsprayed trees).

[7]Lesions $cm^{-2}$ leaf area x $10^{-3}$.

Means followed by the same letter do not differ significantly according to Student-Newman-Keuls Test.

**Significant at p = 0.05. *Significant at p = 0.1.

| Date of 1st spray | Schedule[1] | Unprotected infection periods (#)[2] | Tree phenology[3] | | $Q_o$[4] | Cluster scab | | | % Scabbed fruit | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | Floral | Cluster leaf area ($cm^2$) | | Incidence[5] | Severity[6] | Density[7] | 1 July | 24 Aug. |
| | | | | | McIntosh | | | | | |
| 23 April | A | 0 | ST | 0.0 | 20 | 2.0 a** | 2 | 0.29 a* | 0.0 a** | 1.0 a** |
| 9 May | B | 1 | 1.2 cm GR | 9.6 | 414 | 40.0 b | 59 | 13.75 b | 1.3 ab | 0.3 a |
| 9 May | C | 1 | 1.2 cm GR | 9.6 | 414 | 38.6 b | 56 | 13.05 b | 3.3 b | 2.3 ab |
| 21 May | D | 2 | FS | 17.3 | 566 | 32.0 b | 46 | 8.96 b | 5.0 b | 7.6 b |
| 21 May | E | 2 | FS | 17.3 | 566 | 41.3 b | 70 | 13.64 b | 1.3 ab | 5.3 ab |
| 24 May | F | 3 | FS | 17.3 | 637 | 45.0 b | 42 | 8.49 b | 7.0 b | 6.0 ab |
| Unsprayed | P | 5 | — | 17.3 | 1,253 | 60.0 b | 68 | 19.70 b | 1.6 b | 41.0 c |
| | | | | | Cortland | | | | | |
| 23 April | A | 0 | ST | 0.0 | 20 | 2.0 a* | 2 | 0.29 a** | 0.0 a* | 0.0 a* |
| 9 May | B | 1 | 1.2 cm GR | 9.6 | 414 | 33.3 b | 62 | 10.95 b | 1.3 b | 1.3 ab |
| 9 May | C | 1 | 1.2 cm GR | 9.6 | 414 | 22.6 b | 21 | 5.82 b | 0.0 a | 0.3 a |
| 21 May | D | 2 | FS | 17.3 | 566 | 30.1 b | 34 | 7.21 b | 1.0 b | 2.3 ab |
| 21 May | E | 2 | FS | 17.3 | 566 | 24.0 b | 30 | 5.84 b | 1.3 b | 3.0 ab |
| 24 May | F | 3 | FS | 17.3 | 637 | 32.0 b | 22 | 6.37 b | 2.7 b | 4.3 b |
| Unsprayed | P | 5 | — | 17.3 | 1,253 | 16.0 ab | 20 | 5.78 b | 5.0 b | 13.0 b |

Table 8. The effect of fungicide spray program starting date on terminal scab lesion development on McIntosh and Cortland trees at the Mast Road research orchard, 1985.

[1]From Table 2.

[2]Number of unprotected infection periods prior to the first fungicide application.

[3]Noted for the last infection period prior to the first spray. ST = silver tip; GR = green; FS = fruit set.

[4]Cummulative ascospores $m^{-3}$ air trapped prior to the first spray.

[5]Percentage of terminals with scab.

[6]Number of lesions per 75 terminals (50 terminals for unsprayed trees).

[7]Lesions $cm^{-2}$ leaf area x $10^{-3}$.

Means followed by the same letter do not differ significantly according to Student-Newman-Keuls Test.

$^{***}$Significant at p = 0.01. $^{**}$Significant at p = 0.05. $^{*}$Significant at p = 0.1. ns = not significant.

| Date of 1st spray | Schedule[1] | Unprotected infection periods (#)[2] | Tree phenology[3] | | $Q_o$[4] | Terminal scab | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Floral | Terminal leaf area ($cm^2$) | | 27 June | | | 24 August | | |
| | | | | | | Incidence[5] | Severity[6] | Density[7] | Incidence | Severity | Density |
| | | | | | McIntosh | | | | | | |
| 23 April | A | 0 | ST | 0.0 | 20 | 0.6 a*** | 6 | 0.26 a** | 1.2 a*** | 29 | 0.17 a** |
| 9 May | B | 1 | 1.2 cm GR | 2.1 | 414 | 0.5 a | 8 | 0.23 a | 1.3 a | 19 | 0.15 a |
| 9 May | C | 1 | 1.2 cm GR | 2.1 | 414 | 1.2 a | 17 | 0.48 a | 1.4 a | 28 | 0.53 b |
| 21 May | D | 2 | FS | 47.0 | 566 | 2.9 a | 59 | 1.69 ab | 5.3 ab | 137 | 1.08 c |
| 21 May | E | 2 | FS | 47.0 | 566 | 2.6 a | 100 | 2.52 ab | 7.3 b | 238 | 1.79 c |
| 24 May | F | 3 | FS | 56.2 | 637 | 1.4 a | 18 | 0.51 a | 2.4 a | 94 | 0.74 bc |
| Unsprayed | P | 5 | — | — | 1,253 | 16.5 b | 453 | 20.25 b | 17.1 c | 1,375 | 17.33 d |
| | | | | | Cortland | | | | | | |
| 23 April | A | 0 | ST | 0.0 | 20 | 0.4 a*** | 6 | 0.58 ns | 0.8 a*** | 38 | 0.15 ab |
| 9 May | B | 1 | 1.2 cm GR | 2.1 | 414 | 1.1 a | 34 | 0.78 | 1.5 ab | 28 | 0.20 ab |
| 9 May | C | 1 | 1.2 cm GR | 2.1 | 414 | 0.4 a | 4 | 0.56 | 0.7 a | 12 | 0.09 a |
| 21 May | D | 2 | FS | 47.0 | 566 | 2.2 a | 73 | 3.52 | 2.7 ab | 117 | 0.91 ab |
| 21 May | E | 2 | FS | 47.0 | 566 | 0.5 a | 10 | 3.97 | 3.4 ab | 143 | 1.02 b |
| 24 May | F | 3 | FS | 56.2 | 637 | 1.5 a | 17 | 2.0 | 1.9 ab | 53 | 0.51 ab |
| Unsprayed | P | 5 | — | — | 1,253 | 5.2 b | 91 | 3.83 | 4.9 b | 138 | 0.98 ab |

(schedules A and C). There were no other significant differences in percentage of fruit scab on treated Cortland trees. Thirteen percent of the unsprayed Cortland apples had at least one scab lesion compared to 41% on unsprayed McIntosh trees.

**Foliar scab on immature trees, 1985.** The highest incidence (18-32%) of cluster scab occurred in trees that were left unsprayed until after the first infection period (schedules G-J, Table 9). The amount of cluster scab in these trees was similar, even though trees left unsprayed for each succesive infection period were exposed to more ascospores. A low incidence (<6.6% ) or no cluster scab was recorded on trees that were protected for at least the first infection period (schedules H-L). The highest incidence of scab on terminal leaves occurred on trees unprotected until fruit set on 21 May (schedules H,I,J) or after bloom on 15 May (schedules K,L). With the exception of schedule O, the incidence of terminal scab did not increase from 9 July through 30 August in any schedule.

**Seedling study.** The seedling study was conducted at the Mast Road orchard in 1984 and 1985. Because of the high inoculum density in 1984, benomyl at 3.36kg/ha was applied to reduce the overwintering inoculum. The effectiveness of the post-harvest treatment was reflected in fewer ascospores trapped in 1985. The total ascospores recorded by the Burkhard spore trap was 71,124.2 spores $m^{-3}$ air in 1984 and 1,323.1 spores $m^{-3}$ air in 1985, a 98% reduction in airborne inoculum in 1985. There was also a corresponding reduction in lesion densities on McIntosh seedlings in 1985. Five lesions were recorded

Table 9. Scab incidence on cluster and terminal leaves of immature McIntosh apple trees unprotected with fungicide for different infection periods at the Mast Road research orchard, 1985.

| Schedule | Spray program | | $Qo^2$ | % Foliar scab[1] | | |
|---|---|---|---|---|---|---|
| | First spray | Last spray | | Clusters 9 July | Terminals 9 July | Terminals 30 Aug. |
| G | 9 May | 2 Aug. | 414 | 31.6 | 20.0 | 8.0 |
| H | 22 May | 2 Aug. | 566 | 20.0 | 25.0 | 17.0 |
| I | 24 May | 2 Aug. | 637 | 18.0 | 32.0 | 13.0 |
| J | 30 May | 2 Aug. | 1,061 | 23.3 | 41.6 | 30.0 |
| K | 23 April | 1 May | 1,233 | 6.6 | 28.3 | 12.0 |
| L | 23 April | 15 May | 839 | 5.0 | 23.3 | 12.0 |
| M | 23 April | 22 May | 687 | 0.0 | 15.0 | 8.0 |
| N | 23 April | 22 May | 687 | 0.0 | 10.0 | 10.0 |
| O | 23 April | 4 June | 172 | 0.0 | 1.6 | 6.0 |

[1]% of clusters or terminals with scab.

[2]Total ascospores $m^{-3}$ air recorded by Burkhard trap prior to first spray (G-J) or after last spray (K-O).

on seedlings in 1985 compared to 1,914 lesions in 1984.

Lesion density as a function of ascospore density for both years is presented in Table 10 and Figure 5. The lesion densities for leaves S through -3 include lesions on two leaves too small for measurement during the infection period. A regression of lesion density versus ascospore density for 1984 and 1985 yielded the equation $Y = 1.79 + 0.0073X$ ($R^2 = .86$), where Y is the number of lesions per $cm^2$ leaf tissue and X is the number of ascospores per $m^3$ air trapped by the Burkhard trap.

## Discussion

The experimental approach utilized in this study differed from most previous studies on apple scab in that it attempted to determine the quantity of foliar scab infections that occurred during individual primary infection periods. The main strategy was to leave different trees unprotected for different primary infection periods. In a survey of the literature pertaining to apple scab, the study most closely resembling the present study was conducted by Frey and Keitt (33). They placed paper bags over branches in order to expose different leaf tissue to different infection periods. They were able to identify an interval from May to June during which infections occurred, but presented no quantitative data for disease that occurred during individual primary infection periods. The results of the present study show that the quantity of scab infections that

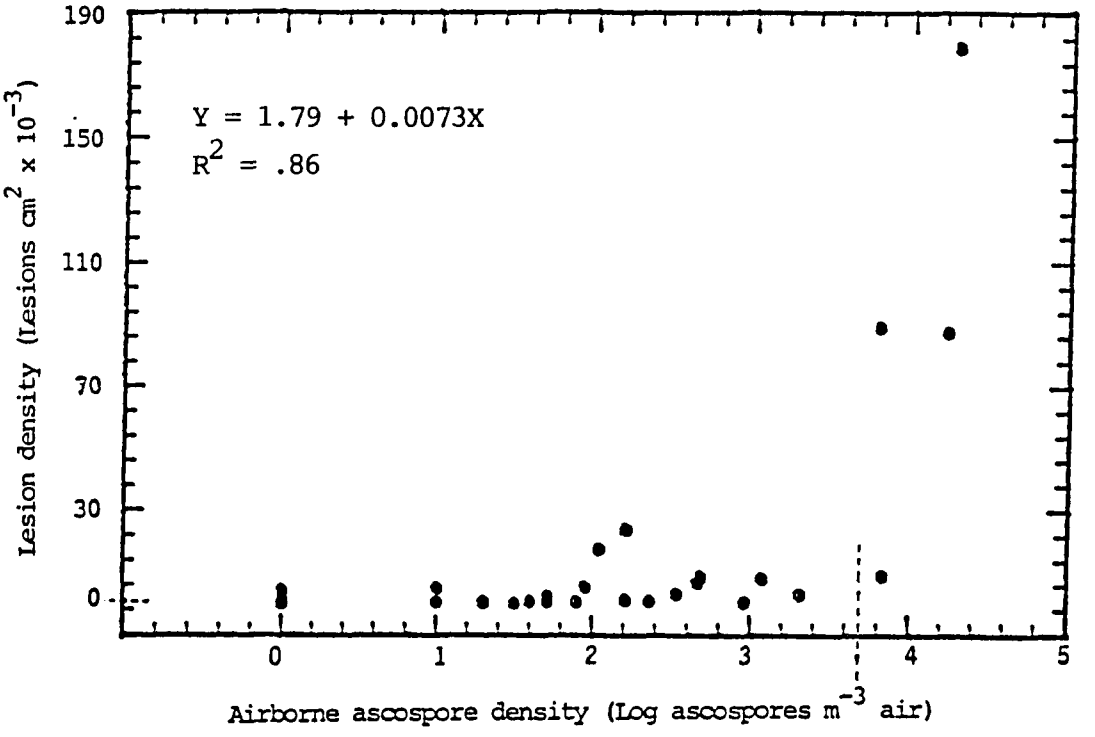



Figure 5. The relationship between airborne ascospore density and lesion density on McIntosh seedlings. Airborne ascospore densities were determined using a Burkhard recording volumetric spore trap. Dashed line represents airborne ascospore density of 5000 ascospores $m^{-3}$, below which there was no relationship between airborne ascospore density and lesion density.

Table 10. The relationship between the density of airborne ascospores of Venturia inaequalis and the density of foliar scab lesions on McIntosh seedlings placed in the Mast Road research orchard. Airborne ascospores were measured with a Burkhard volumetric spore trap.

[1]Date of infection period when seedlings were in the orchard.

[2]Ascospores $m^{-3}$ air trapped while seedlings were in the orchard.

[3]Lesions $cm^{-2}$ leaf area x $10^{-3}$.

[4]Leaf position according to Szkolnik (121) and Fig. 4. S was the string leaf which was the youngest, unfurled leaf when the seedlings were placed in the orchard. Leaves (S) - (-3) included any leaves above the string leaf which developed lesions, the string leaf, and the two leaves below the string leaf. Leaves (-4) - (-7) included the third, fourth, fifth and sixth leaves below the string leaf.

| Date[1] | Airborne ascospore density[2] | Lesion density[3] by leaf position[4] (S)-(-3) | (-4)-(-7) | All leaves |
|---|---|---|---|---|
| 4 May '84 | 19,159.7 | 470.3 | 84.5 | 178.8 |
| 8 May '84 | 16,998.3 | 284.7 | 28.3 | 87.5 |
| 12 May '84 | 6,817.5 | 20.2 | 6.8 | 9.0 |
| 14 May '84 | 6,383.2 | 233.6 | 38.3 | 88.8 |
| 4-5 May '84 | 2,020.0 | 11.2 | 0.0 | 3.0 |
| 28 May '84 | 1,171.6 | 28.0 | 3.2 | 8.0 |
| 12 May '84 | 919.1 | 2.0 | 0.0 | 0.4 |
| 3-4 May '84 | 484.8 | 19.1 | 5.0 | 8.6 |
| 14-15 May '84 | 464.6 | 43.6 | 0.3 | 6.6 |
| 29 May '84 | 343.3 | 6.4 | 2.3 | 3.0 |
| 28 May '85 | 232.3 | 3.2 | 0.0 | 0.8 |
| 13-14 May '84 | 161.6 | 5.9 | 0.0 | 1.1 |
| 28 May '84 | 161.6 | 90.4 | 8.5 | 23.7 |
| 23-24 May '84 | 111.1 | 67.8 | 5.4 | 17.5 |
| 26-27 May '84 | 90.9 | 6.7 | 4.9 | 5.2 |
| 3 May '85 | 80.8 | 1.6 | 0.0 | 0.6 |
| 8-9 May '84 | 50.5 | 9.6 | 1.0 | 2.3 |
| 28-29 May '85 | 50.5 | 2.8 | 0.0 | 0.6 |
| 21-22 May '85 | 40.4 | 2.1 | 0.0 | 0.7 |
| 28-29 May '84 | 30.3 | 0.0 | 0.0 | 0.0 |
| 29-30 May '84 | 20.2 | 0.0 | 0.3 | 0.3 |
| 31 May '84 | 20.2 | 1.5 | 0.0 | 0.3 |
| 12-13 May '84 | 10.1 | 2.7 | 5.3 | 4.7 |
| 30 May '84 | 10.1 | 0.7 | 0.6 | 0.6 |
| 20-21 May '84 | 0.0 | 24.4 | 0.7 | 4.1 |
| 30-31 May '84 | 0.0 | 5.6 | 0.0 | 1.8 |
| 31 May '84 | 0.0 | 1.4 | 0.0 | 0.3 |
| 27 May '85 | 0.0 | 0.0 | 1.0 | 0.8 |
| 30 May '84 | 0.0 | 0.0 | 0.0 | 0.0 |

occurs during an individual primary infection period will depend, to a certain extent, on the point in the primary season when the infection period occurs. Two trends noted were that 1) most cluster lesions were established at the beginning of the primary season, with few or none at the end, and 2) few terminal lesions were established at the beginning of the primary season and most occurred at the end of the primary season.

Several lines of evidence indicate that once cluster leaves reach their maximum size they become highly resistant to scab. In 1984 and 1985, cluster leaves had stopped expanding by petal fall, and in the Woodman Farm study, cluster lesions were only recorded for trees which were unprotected with fungicide for infection periods that occurred prior to or during petal fall. In 1985, no cluster lesions were recorded for trees that were unprotected for infection periods after bloom even though more that 70% of the season's total of ascospores were trapped after bloom. The greatest ascospore/cluster lesion ratio, which was 10 fold larger than any other ratio, occurred in 1984 during an infection period that lasted from petal fall through fruit set. This large ratio suggests that the cluster leaves were highly resistant to scab. Lesions were recorded, however, which points out that although the leaves had become highly resistent to scab, they were not immune. It is possible that the prolonged infection period presented ideal conditions for infection that enabled a small proportion of the deposited ascospores to overcome the increased resistance of the cluster leaves.

Results of the 1985 Mast Road orchard study also indicate that cluster leaves become highly resistant to scab when they attain their

maximum size. McIntosh and Cortland trees unprotected for the first two, first three, or all five primary infection periods had incidences of cluster leaf scab similar to trees unprotected for only the first infection period. These similarities could not be attributed to exposure to the same amount of airborne inoculum, as 67% of the season's total of ascospores were trapped after the first infection period. However, the cluster leaves ceased expansion between the first and second primary infection periods. A similar pattern was observed on immature trees, where the greatest incidence of cluster lesions were observed on trees unprotected for the first infection period. Less than 6.6% of the clusters on trees unprotected for infection periods after petal fall were infected.

The greatest lesion densities on terminal leaves were associated with infection periods that occurred toward the end of the primary season, a pattern opposite that detected on cluster leaves. In the 1985 Woodman Farm study, terminal lesion densities ranged from 0 to $4.0 \times 10^{-3}$ lesions $cm^{-2}$ susceptible leaf area, with 0 recorded for the first two primary infection periods and the largest density recorded for the final primary infection period. The low terminal lesion densities recorded for the first primary infection periods were attributed, in part, to the sparse amount of susceptible terminal leaf tissue during infection periods prior to pink.

The terminal lesions recorded in 1985 in the Woodman Farm study were likely caused by infections by ascospores, but terminal lesions recorded in all plots in 1984 may have been caused by infections by ascospores and conidia. This was because the lengthy infection period on 28 May - 2 June that ended the 1984 primary scab season resulted in

secondary infections which obscured the identification of lesions that developed from previous infection periods. At the beginning of the wet period, trees in plots A, B, and C were protected with captan that had been applied on 25 May. By 30 May, it was likely that sufficient rain (9.5 cm) had fallen to reduce the captan residue below the proposed threshold level of 1-2 ug $cm^{-2}$ necessary for scab control (108). Thus, from 30 May to 2 June, the captan residue was insufficient in these plots to protect against secondary infections. The primary lesions in those plots which had been initiated earlier in the growing season could then provide a source of conidia within the tree canopies.

There are several lines of evidence to suggest that many terminal lesions recorded for plots A, B, and C in 1984 were caused by conidia. In plot A, terminal lesions were recorded even though terminal leaves had not begun to grow when these trees were unprotected. Likewise, terminal lesions in plot B were likely a mixture of primary and secondary lesions. Terminal lesions were recorded on leaves 4-10, but only six leaves per terminal were present when the trees were unprotected. In plot C, evidence for secondary infections includes the presence of sheet scab on some leaves and lesions recorded on leaves 13 and 14 which were not present on 12-14 May, when the plot was unprotected with fungicide. Based on available information on the latent period for V. inaequalis (127), infections that occurred on 12-14 May would likely have developed into sporulating lesions between 28 May and 1 June. Thus, it appears that some terminal lesions in plot C resulted from secondary infections.

The presence of sheet scab on trees in plot D on 1 July raises a

question as to the origin of the inoculum causing the sheet scab. Sheet scab is believed to be caused by conidia that are splash-dispersed, causing multiple infections and lesions on the leaf from which they originate or on other leaves throughout the tree canopy. These lesions are so numerous as to lose their individual identity. Assuming that the observed sheet scab was caused by conidial infections, the inoculum would have originated within the tree canopies where the lesions were observed, or would have been dispersed from lesions in other plots. Evidence for the former explanation comes from 6 years of spore trapping in the present study and with a Burkhard spore trap at the high-inoculum Mast Road research orchard where no conidia of V. inaequalis have ever been trapped by traps placed outside a tree canopy during the primary season. Others (21) have also contended that conidia are not important in the spread of scab over distances beyond an adjacent tree. The former explanation also implies that fungicide sprays are not 100% effective, as plot D was sprayed on a weekly basis until 13 May. This is not an unreasonable argument. For example, trees sprayed throughout the primary season on a 7-10 day protectant schedule at the Mast Road research orchard in 1985 had 2% cluster scab when indexed on 27 June. The latter explanation implies dispersal of conidia beyond the canopy of the tree in which they originate, a phenomenon which needs to be studied more carefully. Conidia have been trapped beyond the canopy by Howitt (57), who trapped conidia on vaseline coated slides up to 12 m from trees with lesions on 12 June, and up to 22 m on 20 July, and by Allit (3), who trapped conidia from the roof of a hospital located in the city of Cambridge, UK.

There is no evidence from this study that the early development of scab lesions on cluster leaves contributed significantly to fruit scab in an orchard with proper fungicide protection later in the growing season, although cluster lesions are close to the developing fruit and are a source of conidia. Cluster lesions were extensive at the Mast Road orchard in 1985. One or more scab lesions developed on 32 to 45% of the clusters in all trees except trees protected throughout the primary season. Yet, the maximum percentage of scabbed fruit recorded on 24 Aug. was only 7.6% on McIntosh and 4.3% on Cortland trees sprayed with fungicide throughout the secondary season. Less than 25% of the clusters with scab lesions contained scabbed fruit, and scabbed fruit often occurred within clean clusters. At the Woodman Farm, the plot with the most cluster lesions in 1984 had the lowest percentage of scabbed fruit. Thus, lesions on cluster leaves, though close to developing fruit, did not appear to produce conidia that contributed significantly to the development of scabbed fruit. Jeger (60) was also unable to utilize leaf scab intensity or incidence to satisfactorily predict fruit scab intensity.

These findings are in contrast to those of Keitt and Jones (64) who noted that fruit scab incidence was greatest when infections occurred on sepals and cluster leaves early in the season. However, they were examining fruit on trees unsprayed during the entire growing season. Dale et al. (23), over a four year period, found that delaying the start of a fungicide program led to an increase in the amount of fruit scab at harvest. In their study, the percentages of scabbed fruit from spray programs that began approximately one month apart were 98.8 vs 97.5%, 95 vs 91.8%, 84.3 vs 55.8%, and 57.8 vs

27.3% in 1963 - 1966, respectively. They attributed the decreased level of control each year to increased inoculum, inadequate pruning which limited fungicide coverage, leaching by frequent rain in the latter years, and reduction of fungicide rate. However, they applied dodine exclusively in the orchard, and had been using dodine in that orchard for at least three years prior to the study (21). Because the development of tolerance to dodine is common with repeated use of dodine (40,82,123), the data of Dale et al. probably reflected a build-up of dodine tolerance in the experimental orchard during the course of their study. If true, then the study did not accurately assess the effect of fungicide spray starting date on final percentages of fruit scab, with the possible exception of 1963.

In the 1985 Mast Road study, fruit scab on trees unprotected until after bloom was greater than on trees sprayed at least once prior to bloom, but the highest incidence of fruit scab on 24 August was only 7.6% on McIntosh and 4.3% on Cortland despite 22 secondary infection periods. These percentages are above tolerance levels of most commercial growers, but they do indicate that fruit scab incidence may not increase dramatically with a reduction in fungicide applications during the primary scab season. Trees in the protectant schedule received 11 fungicide sprays compared to 7 sprays for trees first treated at fruit set, but only one of the three schedules in the latter group (schedule D on McIntosh, schedule F on Cortland) had significantly ($p = 0.05$) more fruit scab. A slight increase in fruit scab could be justified and tolerated by a grower interested in marketing fruit advertised as being produced with a minimum number of chemical applications. Such a marketing strategy could be profitable

in a carcinophobic society where people might conceivably pay the same price or more for apples with a higher incidence of scab if produced with fewer sprays.

Dodine is a sporulation inhibitor (2), but there was no apparent inhibitory effect in this study. Because the development of some foliar lesions was expected, dodine had been included in some of the spray schedules to determine if limiting conidia production in those lesions would lessen the build-up of secondary scab and fruit scab throughout the growing season. On McIntosh trees first sprayed at pink, the dodine treated trees had significantly ($p = 0.05$) more terminal scab than their dodine free counterparts (B), but no statistically significant differences were noted from Cortland trees sprayed with maneb or dodine at pink. Similarly, there were no significant differences in terminal scab between McIntosh or Cortland trees first treated with dodine or maneb at fruit set. There were no statistically significant differences in the percentage of scabbed fruit between any dodine and dodine-free schedules on 1 July or 24 Aug.

The inoculum source in the Woodman Farm research plots was much greater than would be expected in a well-managed commercial orchard with <5% foliar scab. Gadoury (38) estimated that an orchard with 4.5% foliar scab would have 42.2 lesions $m^{-2}$ orchard floor, and an orchard with 20% leaf scab would contain 205 lesions $m^{-2}$ orchard floor. The point source of inoculum in the research plots was estimated in Chapter III to contain 2500 lesions $m^{-2}$ of orchard floor. However, despite the large inoculum source, no consistent relationship was determined between airborne ascospore densities recorded at the

source or in the tree canopies and cluster or terminal lesion densities in any plot. The difficulty in establishing a relationship was due to the low numbers of lesions observed in each plot and not to any inherent inefficiency of the volumetric spore traps to accurately detect low airborne ascospore densities.

Closer examination of the relationship between airborne ascospore density and lesion density established with seedlings at the Mast Road orchard (Fig. 5) helps to explain the inconsistent relationship recorded for the Woodman Farm study. Only when the airborne ascospore density was greater than 5000 ascospores $m^{-3}$ air was there a relationship with lesion density. Below 5000 ascospores $m^{-3}$ air, the relationship between airborne ascospore density and lesion density was inconsistent, with a maximum of $23.0 \times 10^{-3}$ lesions $cm^{-2}$ leaf area recorded. In the two years the Woodman Farm study was conducted, the maximum ascospore density measured at the source by the 7-Day recording volumetric spore trap was 482 ascospores $m^{-3}$ air, and the airborne ascospore densities in the canopies 2-6 m from the source were much less. Thus, for every infection period in 1984 and 1985, the density of airborne ascospores at the Woodman Farm would have been at the lower end of the curve in Figure 5, explaining the inconsistencies when trying to relate airborne ascospore density to lesion density at the Woodman Farm.

It may be that the plot size in the Woodman Farm study was not large enough to detect a low threshold of lesions that was developing. However, to increase the plot size by adding more trees would have resulted in some trees being farther away from the inoculum source, where fewer ascospores would be available to cause infections. Also,

although data were only reported for 50 cluster and 50 terminals per tree, many more were examined to ensure that the low number of lesions being recorded was an accurate assessment of the amount of disease in the entire tree.

Hirst and Stedman (53) stated that the initial infection in an unsprayed orchard would be proportional to the concentration of airborne ascospores, but they presented no data to substantiate their claim. Results from the present study indicate that the amount of initial infection will be proportional to the airborne ascospore density only when there are exceptionally large amounts of inoculum in an orchard. Usually, such inoculum levels are found only in abandoned orchards or in plant pathology research orchards.

In commercial orchards that contain much less inoculum, i.e., well below 2500 lesions $m^{-2}$ orchard floor, the relationship between airborne ascospore density and lesion density can not be precisely defined. However, some generalizations may be made about lesion densities that might occur in a low inoculum commercial McIntosh or Cortland orchard with insufficient or no fungicide coverage during a primary infection period. For infection periods that occur prior to pink, infections would be limited to the cluster leaves with lesion density not expected to exceed $1.5 \times 10^{-3}$ lesions $cm^{-2}$ cluster leaf tissue. From pink through petal fall, lesions may occur on cluster and terminal leaves. The range expected in cluster lesion density would be the same as above. Terminal lesion density in the present study did not exceed $59.3 \times 10^{-3}$ lesions $cm^{-2}$ terminal leaf tissue, but the largest values included primary and secondary lesions. In the absence of conidia, a more conservative range of $0\text{-}23.0 \times 10^{-3}$ lesions

$cm^{-2}$ might be expected, the upper limit based on the maximum lesion density recorded on seedlings when the airborne ascospore density was less than 5000 ascospores $m^{-3}$ air. After petal fall, new lesions would be restricted to the highly susceptible youngest seven terminal leaves, and few, if any, lesions would be expected to develop on older leaves under low inoculum conditions. If scabbed leaves are present at that time, the terminal lesion density on unprotected tissue might approach densities of $59.3 \times 10^{-3}$ lesions $cm^{-2}$ recorded in 1984. Although missing a fungicide application in the primary scab season will result in an increase in the amount of foliar scab, there should be no effect on final amounts of fruit scab if the grower makes careful and timely fungicide applications after the missed application.

A major factor that limits the number of primary infections early in the growing season is the small amount of susceptible tissue that an ascospore can impinge upon. The ascospore/lesion ratios measured in the Woodman Farm study were greatest for cluster lesions initiated at the beginning of the primary season, less for cluster lesions just prior to the attaining their maximum size, and smallest for terminal lesions initiated later in the growing season. The decrease in the ascospore/lesion ratio as the tree canopy filled in may have been the result of a greater deposition efficiency for V. inaequalis ascospores when more foliage was present. Carter (18) showed that the deposition of ascospores by Eutypa armeniaceae increased as the amount of apricot foliage increased. The mechanism of ascospore discharge and dispersal during rainy periods are similar to those of V. inaequalis.

Low numbers of lesions resulting from primary infection periods

can be explained by examining the life history of V. inaequalis. Through evolution, in the absence of fungicides, the fungus has developed mechanisms of spore production and release which ensure that ascospores are present in the atmosphere at a time most conducive for infection, i.e. when it is raining and young, susceptible leaf tissue is present. Only a few lesions actually develop from infection periods occurring early in the growing season, but these lesions produce conidia that can cause secondary infections. The splash-dispersal of conidia within the tree canopy throughout the growing season is an evolutionary characteristic which is responsible for establishing high lesion densities. Large numbers of lesions are necessary because many scabbed leaves fall and decay prior to the onset of winter, and the population size at leaf fall must be sufficient for insuring successful overwintering of the pathogen and renewed contact with the host the following spring. The build-up of scab throughout the growing season also increases the probability that opposite mating types will make the contact necessary to produce the overwintering, sexual stage of the fungus.

The few lesions that result from primary infections suggest more flexibility in scheduling early season sprays. It may be possible to omit sprays until the appearance of secondary inoculum. Fungicides available today have shown considerable effectiveness in limiting lesions under high inoculum pressure maintained in research trials (104,105,106,121), to the extent that a spray program based on the first appearance of secondary inoculum may be as effective as a program designed to prevent all initial infections. Under such a program, the first fungicide application might be applied at the pink

bud stage. In New Hampshire, the first primary lesions are usually not initiated until 0.6 - 1.3 cm (1/4 - 1/2") green. In the past 7 years, the average interval between the 1.3 cm green and pink bud stages has been 13 days. Tomerlin and Jones (127) have reported that at least 10 days are required before secondary inoculum is produced. The latent period is longer at the cooler temperatures which prevail in New Hampshire at the beginning of the primary season, so in most years secondary infections would not occur until pink. Lesions prior to pink will be restricted to cluster leaves, but their presence should not contribute to an unacceptable incidence of fruit scab at harvest. Pink is a convient floral growth phase to start a scab control program, because growers usually apply an insecticide for tarnished plant bug at pink. Thus, both pesticides could be applied simultaneously, saving the grower time and money. Delaying the first fungicide spray for scab until pink would result in one, two, or even three fewer sprays during the primary scab season.

One negative aspect of delaying the first fungicide spray would be an increase in the amount of foliar scab and, thus, an increase in overwintering inoculum. However, techniques have been developed to estimate the amount of overwintering inoculum in an orchard based on the amount of foliar scab at the end of the growing season (38), and the effectiveness of a post-harvest fungicide applications in reducing the overwintering inoculum has been shown here and in other studies (37). If the build-up of scab in a commercial orchard exceeded a tolerance level for overwintering inoculum, a single post-harvest application could reduce the overwintering inoculum to an acceptable level. A post-harvest spray would actually be an additional spray

because growers do not apply fungicide sprays after harvest. However, over time, the occasional post-harvest application would still result in less fungicide applied each year than is presently being applied in pre-pink sprays for control of apple scab in New Hampshire. Other, non-pesticidal chemical means could also be used to reduce overwintering inoculum, e.g. urea applications or sanitation practices such as shredding or removing leaves.

Studies on apple scab usually represent lesion data as the proportion of leaves with scab (27,123,136), but expressing disease as lesion density provides a more accurate indication of the lesions present in the orchard. For example, there was no significant difference between plots C and E (Table 4) on 1 Sept. in the percentage of terminal leaves with lesions, but the lesion density (and number) was significantly greater in plot E (Table 6). Such differences may be critical, especially if management decisions are influenced by the amount of scab present at the end of the primary scab season. With a procedure for forecasting the overwintered ascospore dose (38), a grower has the option to adjust his/her scab management program based on the inoculum level. A post-harvest measure to reduce an inoculum dose that is above a specific threshold is one adjustment, and adjusting the starting date of a spray program the next year based on inoculum dose below a specified threshold is another. The effectiveness of scab management decisions will be increased by expressing disease in a manner which best highlights differences in the amount of scab in an orchard.

# Chapter V.

## GLASSHOUSE STUDIES ON THE RELATIONSHIP BETWEEN AIRBORNE ASCOSPORE DENSITY AND DEPOSITED ASCOSPORE DENSITY IN *VENTURIA INAEQUALIS*

### Introduction

The previous chapter examined the development of scab lesions on apple trees which were unprotected with fungicide and located adjacent to a large inoculum source. Volumetric spore traps located within the tree canopies trapped ascospores during most rainy periods. Average ascospore densities of 0.001 to 0.09 spores $m^{-3}$ air were measured in the tree canopies, but few lesions developed. Two possible explanations for the low number of scab lesions are; 1) the proportion of airborne ascospores actually deposited on susceptible leaf tissue was small relative to the airborne ascospore density; or 2) only a small proportion of deposited ascospores were capable of infecting the apple leaf. The purpose of this study was to investigate the first explanation by determining in a glasshouse the relationship between airborne ascospore density and deposited ascospore density.

## Materials and Methods

The study was conducted at the University of New Hampshire glasshouse facilities in Durham. An inoculum source of approximately 200 leaves infected with V. inaequalis was obtained from unsprayed McIntosh check trees at the Mast Road Research Orchard in October, 1984, placed in a wire cage, and overwintered in that orchard. On April 1, 1985, when pseudothecia were developed but not yet containing mature ascospores, the leaves were placed in a freezer and stored until experiments were conducted in September, 1985. Prior to commencement of the experiment, the leaves were placed in a growth chamber at 10 C to induce ascospore maturation.

The glasshouse was approximately 4 x 8 m and contained two benches, each 1.5 x 7 m. One bench contained spruce and fir seedlings approximately 0.5 m tall. The cage with the infected leaves was placed at one end of the second bench (Fig. 1). Six volumetric spore traps (Appendix A) were placed on the bench, two each at distances of 0.9, 1.8 and 2.7 m from the infected leaves. A McIntosh apple seedling was placed directly in front of each spore trap below the level of the trap intake. One 22 $mm^2$ coverslip was appressed with petroleum jelly to each of two leaves on each seedling. One coverslip was placed on the upper leaf surface of the youngest, expanded leaf. The seedling was adjusted so that this coverslip was facing the ascospore source. The second coverslip was placed on the upper surface of the second youngest leaf. The lower surface of this leaf was facing the inoculum; thus, the coverslip was oriented away from the inoculum source (Fig. 2). Both coverslips were at approximately

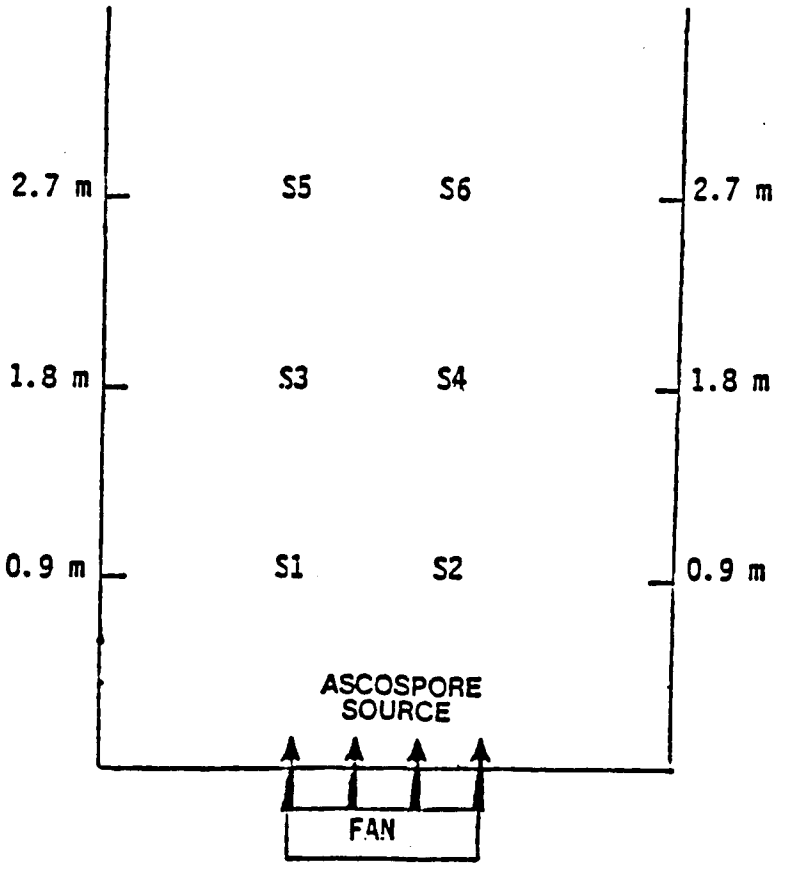



Figure 1. Location of spore traps and seedlings in relation to the ascospore source and wind source on a glasshouse bench. S1-S6 = sites 1-6. Each site contained one volumetric spore trap and one McIntosh seedling.

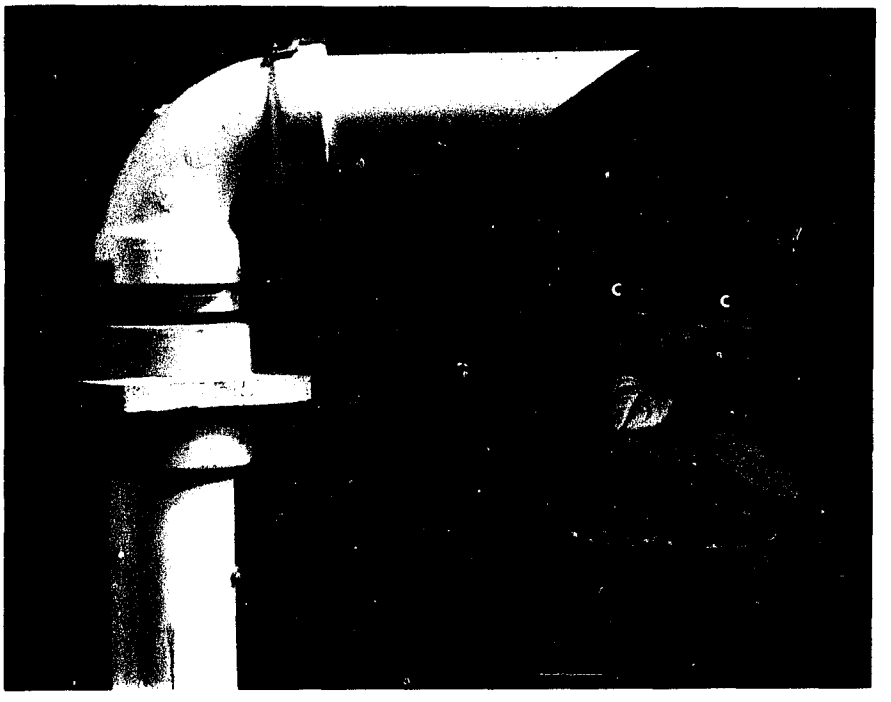



Figure 2. Spore trap and seedling positions at each site on glasshouse bench. Coverslip (C) on right, uppermost leaf was facing away from the inoculum and wind sources; coverslip on left, uppermost leaf was facing the wind and inoculum.

45° angles to the surface of the bench.

A portable fan placed directly behind the spore source provided a wind source (Fig. 1.) Prior to commencement of the experiment, a recording anemometer (WeatherMeasure Corp. Sacremento, CA 95841) was used to determine the wind speed at each site. Wind speed ranged from 0.2 to 0.9 m $s^{-1}$.

Ascospores were released five times over a five week period. Spores were released naturally. Tap water was initially poured directly onto the infected leaves, which were then misted to keep them wet for the duration of each test. All tests were conducted between 0900 and 1300 h in deference to the daily pattern of ascospore release exhibited by V. inaequalis (78). After each release, the leaves were returned to the growth chamber and incubated at 10 C to allow more ascospores to mature. At the completion of each test, the coverslips were removed from each leaf, inverted, and placed on a microscope slide containing several drops of Gelvatol:glycerol:water (35 g:100ml:50ml). Spores on the spore trap slides were mounted in the same solution, and the densities of deposited ascospores $cm^{-2}$ and airborne ascospores $m^{-3}$ were determined, respectively, by scanning the entire coverslip or the entire band of deposited spores, using a compound microscope.

## Results and Discussion

The range of airborne ascospore densities for the five releases was 1.3 to 121.1 ascospores $m^{-3}$ (Table 1), with a minimum of 6 and a maximum of 659 ascospores trapped. Air currents in the glasshouse were such that at each distance from the inoculum source, more ascospores were always trapped at the even numbered site than at its odd counterpart.

The deposited ascospore densities on the coverslips were low, ranging from 0.0 to 0.52 ascospores per $cm^2$ (Table 1). Only 24 ascospores were deposited during the entire study, with 10 recorded in the first release when the most airborne ascospores (1704) were trapped. Seventeen (71%) of the deposited ascospores were on coverslips facing the wind and inoculum source. The most ascospores deposited on a single coverslip was 4. In all other instances, 1 or 2 ascospores were deposited. Seventy percent of the coverslips had no ascospores deposited on them.

Attempts were made to relate airborne ascospore density to deposited ascospore density, using regression analysis with untransformed and transformed data, but there was poor correlation between the two variables. The most ascospores were deposited during the first test. In the remaining four tests there was considerable variation in the relationship between airborne and deposited ascospore density, and the largest airborne spore densities did not always result in the largest deposited densities. An analogy might be drawn between this study and the seedling study described in Chapter IV. In

Table 1. Airborne and deposited ascospore density, area dose, and efficiency of deposition of ascospores of *Venturia inaequalis* in glasshouse studies.

[1] $m\ s^{-1}$

[2] Ascospores $m^{-3}$ air 5 cm above seedling calculated from spores trapped by a volumetric spore trap.

[3] Ascospores $cm^{-2}$ deposited on coverslips placed on the upper and lower surfaces of leaves of apple seedlings. Facing and away describe orientation of coverslip to the inoculum source.

[4] Area dose (Ascospores $cm^{-2}$) = ascospores $m^{-3}$ x wind speed ($m\ s^{-1}$) x duration (s) of spore release.

[5] Percentage of the area dose above a seedling (as determined with a volumetric spore trap) which is deposited on the seedling (as determined with a cover slip).

| Site | Distance (m) from inoculum | Wind velocity[1] | Ascospore Density Airborne[2] | Deposited[3] Facing | Away | Mean | Area Dose[4] | Deposition Efficiency (%)[5] |
|---|---|---|---|---|---|---|---|---|
| | | | Test 1 | | | | | |
| 1 | 0.9 | 0.45 | 19.4 | 0.83 | 0.2 | 0.52 | 12.57 | 4.1 |
| 2 | 0.9 | 0.89 | 121.1 | 0.41 | 0.0 | 0.2 | 155.20 | 0.1 |
| 3 | 1.8 | 0.45 | 35.2 | 0.0 | 0.2 | 0.1 | 22.80 | 0.4 |
| 4 | 1.8 | 0.45 | 65.8 | 0.41 | 0.0 | 0.2 | 42.63 | 0.5 |
| 5 | 2.7 | 0.23 | 15.3 | 0.0 | 0.0 | 0.0 | 5.06 | 0.0 |
| 6 | 2.7 | 0.23 | 55.0 | 0.0 | 0.0 | 0.0 | 18.21 | 0.0 |
| | | | Test 2 | | | | | |
| 1 | 0.9 | 0.45 | 2.2 | 0.0 | 0.2 | 0.1 | 1.07 | 9.3 |
| 2 | 0.9 | 0.89 | 10.0 | 0.0 | 0.2 | 0.1 | 9.61 | 1.0 |
| 3 | 1.8 | 0.45 | 2.7 | 0.0 | 0.0 | 0.0 | 1.31 | 0.0 |
| 4 | 1.8 | 0.45 | 9.7 | 0.0 | 0.0 | 0.0 | 5.49 | 0.0 |
| 5 | 2.7 | 0.23 | 2.2 | 0.0 | 0.0 | 0.0 | 0.54 | 0.0 |
| 6 | 2.7 | 0.23 | 4.7 | 0.0 | 0.0 | 0.0 | 1.16 | 0.0 |
| | | | Test 3 | | | | | |
| 1 | 0.9 | 0.45 | 3.2 | 0.2 | 0.0 | 0.1 | 1.81 | 5.5 |
| 2 | 0.9 | 0.89 | 10.7 | 0.41 | 0.2 | 0.3 | 11.99 | 2.5 |
| 3 | 1.8 | 0.45 | 5.7 | 0.41 | 0.0 | 0.2 | 3.23 | 6.2 |
| 4 | 1.8 | 0.45 | 7.4 | 0.2 | 0.0 | 0.1 | 4.19 | 2.4 |
| 5 | 2.7 | 0.23 | 4.1 | 0.0 | 0.0 | 0.0 | 1.18 | 0.0 |
| 6 | 2.7 | 0.23 | 5.7 | 0.0 | 0.0 | 0.0 | 1.65 | 0.0 |
| | | | Test 4 | | | | | |
| 1 | 0.9 | 0.45 | 3.6 | 0.0 | 0.0 | 0.0 | 2.04 | 0.0 |
| 2 | 0.9 | 0.89 | 49.8 | 0.0 | 0.0 | 0.0 | 55.84 | 0.0 |
| 3 | 1.8 | 0.45 | 3.8 | 0.0 | 0.0 | 0.0 | 2.15 | 0.0 |
| 4 | 1.8 | 0.45 | 33.8 | 0.0 | 0.0 | 0.0 | 19.16 | 0.0 |
| 5 | 2.7 | 0.23 | 1.3 | 0.0 | 0.2 | 0.1 | 0.37 | 26.5 |
| 6 | 2.7 | 0.23 | 3.4 | 0.0 | 0.0 | 0.0 | 0.98 | 0.0 |
| | | | Test 5 | | | | | |
| 1 | 0.9 | 0.45 | 7.1 | 0.0 | 0.0 | 0.0 | 3.73 | 0.0 |
| 2 | 0.9 | 0.89 | 19.5 | 0.0 | 0.2 | 0.1 | 20.30 | 0.5 |
| 3 | 1.8 | 0.45 | 20.0 | 0.0 | 0.0 | 0.0 | 10.53 | 0.0 |
| 4 | 1.8 | 0.45 | 13.7 | 0.2 | 0.0 | 0.1 | 7.21 | 1.4 |
| 5 | 2.7 | 0.23 | 4.6 | 0.2 | 0.0 | 0.1 | 1.23 | 8.1 |
| 6 | 2.7 | 0.23 | 9.1 | 0.2 | 0.0 | 0.1 | 2.44 | 4.0 |

that study, only with extremely high airborne ascospore densities was there a correlation between airborne ascospore and lesion densities. Although airborne ascospore densities in the glasshouse studies were probably above those encountered in commercial orchards, they were apparently below a threshold where a mathematical relationship could be established between airborne and deposited ascospore density.

Kerr and Rodrigo (66) found positive correlation between airborne and deposited spore densities of Exobasidium vexans, the causal agent of blister blight on tea. They were able to predict disease incidence based on airborne spore concentrations. Several features of the blister blight system which aided their study are not applicable in the apple scab system. They were working with a perennial crop in which susceptible tissue and inoculum were present year around. Also, the basidia originated in lesions on the leaves, and were already within the tea canopy. With apple scab, the ascospores originate from the orchard floor and must be carried by the wind to the canopy. Because deposition from and thinning of a spore cloud is greatest near the inoculum source, deposition on foliage of spores that originate within the canopy will be much greater compared to the deposition of spores that originate outside the canopy. Kerr and Rodrigo (66) were also able to wash basidia off leaves allowing them to sample a greater surface area. Such a technique would not work with V. inaequalis ascospores which are very difficult to remove from a surface.

Most deposition studies have utilized very large numbers of spores or particles. Spores of Lycopodium sp. released in large numbers have been utilized to study dispersal and deposition over a grassy field (47) and in a barley crop (9). Fluorescent tracers have

been used to examine spore dispersal and deposition in a barley crop (87), a bean field (112), and a wheat field (113), and to examine patterns of deposition on crops (55). The ease with which large numbers of these particles or spores can be released, trapped, and counted has been the primary factor in their extensive use in dispersal and deposition studies.

In contrast, few deposition or dispersal studies have utilized the spores of fungal pathogens, in part because it is difficult to release then in sufficient numbers. Edmonds and Driver (25) examined the dispersal and deposition of Fomes annosus spores in a forest. When concentrated conidia were released, there was poor correlation between the airborne spore density, determined with Rotorod samplers, and spore deposition on petri plates. Deposition rates from a natural population of F. annosus varied from 0.0003 to 0.007 spores $cm^{-2}$ per hour over a 24 hour period, much lower than the deposition recorded here for V. inaequalis.

A study similar to that described here was conducted by Carter (18) who studied the deposition of ascospores of Eutypa armeniaceae in a wind tunnel. He utilized the concept of area dose, which can be defined as the number of spores passing through an imaginary frame of unit area cross-section at right angles to the wind (46). Airborne ascospore densities were determined using a Cascade Impacter, and area dose was calculated using the formula,

$$\text{Area dose} = \text{spores } cm^{-3} \times \text{wind speed } (cm\ s^{-1}) \times \text{time (s)}.$$

He then determined the efficiency of deposition (E), expressed as the percentage of the area dose deposited per square centimeter of surface. The efficiency of deposition on leaf, petiole and stem

sections, at wind speeds of 1-5 m $s^{-1}$, ranged from 0.05 to 2.79%, with greater efficiency at higher wind speeds. The presence of foliage also increased deposition.

The efficiency of deposition (E) for V. inaequalis ascospores calculated using Carter's method, ranged from 0.1 to 26%, with an average of 3.8, 2.5, and 0.8% at wind speeds of 0.23, 0.45, and 0.89 m $s^{-1}$. Thus, the efficiencies of deposition for ascospores of V. inaequalis and E. armeniaceae are comparable. The decreasing value of E with increased wind velocity in this study is contrary to the findings of others (18,55), but is probably due to the experimental design and not to unique properties of V. inaequalis. In this study, wind velocity varied with respect to position of the traps from the source, so that during each ascospore release, deposition was recorded at several wind velocities. There was only one trap sampling at the highest wind velocity (0.89 m $s^{-1}$), but there were three and two traps at 0.45 and 0.23 m $s^{-1}$, respectively. The highest wind velocity occurred closest to the inoculum source, and, not surprisingly, the highest airborne ascospore densities were also recorded there during each test. Because there was no relationship between airborne ascospore density and deposited ascospore density (and no difference in deposition density regardless of the airborne ascospore density), deposition at higher airborne ascospore densities will seem less efficient than at lower airborne spore densities. However, when the coverslips with at least 1 ascospore were compared, there was a greater percentage of deposition at the highest wind speed (Fig. 3).

The data indicate that a low proportion of airborne ascospores are deposited on foliage. Thus, the low number of lesions recorded

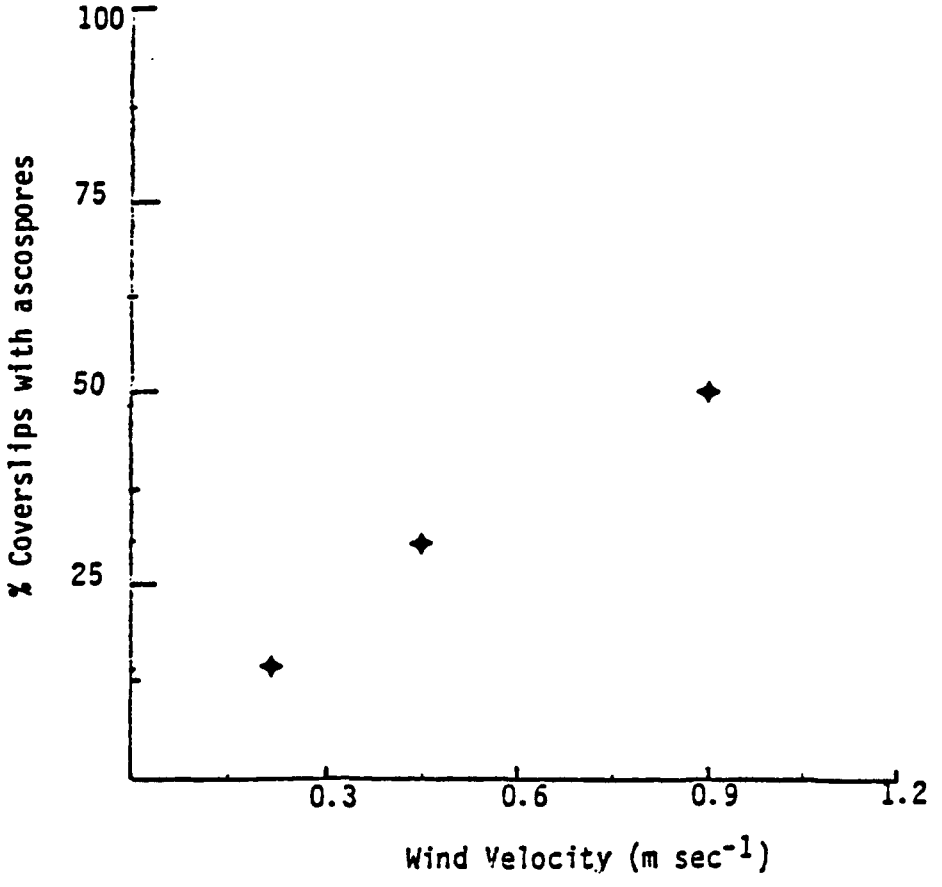



Figure 3. Relationship between wind velocity and the percentage of coverslips with deposited ascospores of Venturia inaequalis in a glasshouse.

in the field studies described in Chapter IV can be explained, at least in part, by the low deposition efficiency of V. inaequalis ascospores. Airborne ascospore area dosages were higher in the glasshouse than in the orchard, and at any point in a well managed commercial orchard, the airborne ascospore density would rarely, if ever, be as high as those recorded in field studies described in Chapter IV. Thus, lesion densities resulting from primary infection periods in commercial orchards would be less than or equal to those recorded in Chapter IV.

Carter (18) noted that deposition of E. armeniacae ascospores was more efficient when more foliage was present, and it would be reasonable to assume that the same would be true of V. inaequalis ascospores which are released and dispersed in a similar manner. Increased efficiency of deposition by ascospores as the amount of apple foliage increases throughout the primary season supports the results of field studies presented in Chapter IV. Those studies showed greater lesion densities (and numbers) resulting from infection periods which occurred at the end of the primary scab season versus those at the beginning. However, the increased lesion densities at the end of the primary scab season did not result from an increase in airborne ascospore densities, as lower airborne ascospore densities were often measured at the end of the primary scab season.

# Appendix A

## Volumetric spore trap used to sample airborne spore density in orchard and glasshouse studies

The spore trap used in these studies is shown in Figure 1. The trap was made of Polyvinyl chloride (PVC), with the exception of the aluminum wind vane (V) and the plastic molding (M) which housed the orifice and glass slide on which spores were deposited (Fig. 2). A Fafner 9806K bearing (Bearings Inc., Dover, NH 03820) mounted inside a 2" x 1" reducing bushing (B, Fig. 1) allowed the top portion of the spore trap to rotate with the wind, keeping the trap intake (I, Fig. 1) facing the wind. The bushing had been bored out to the outer diameter of the bearing.

Attached to the base of each trap was a 1" x 1" PVC coupler (C, Fig. 1) which enabled mounting of the trap onto 1" PVC tubing. The length of the tubing could be adjusted depending upon the desired height of the trap intake.

Air was drawn through the trap orifice with a 12 volt DC blower motor (Mt, Fig. 2) (Herbach and Rademan, Philadelphia, PA 19134) powered as described in Appendix B. Spores were deposited on a glass slide (S, Fig. 2) in a thin band corresponding to the orifice size. The slide rested in a fitted depression in the plastic molding (M).

Each trap was tested in the laboratory to determine the amount of air drawn at 5.0, 5.5, 6.0, 6.5, 7.0, 7.5, and 8.0 volts DC, and within this range, the relationship between the voltage and the amount

of air drawn was linear. When the spore traps were placed in the field, the amount of current reaching each trap was measured with a voltimeter, and the exact amount of air drawn by each trap was calculated. The amount of air sampled by each trap ranged from 17.5 to 19.5 l air $min^{-1}$.

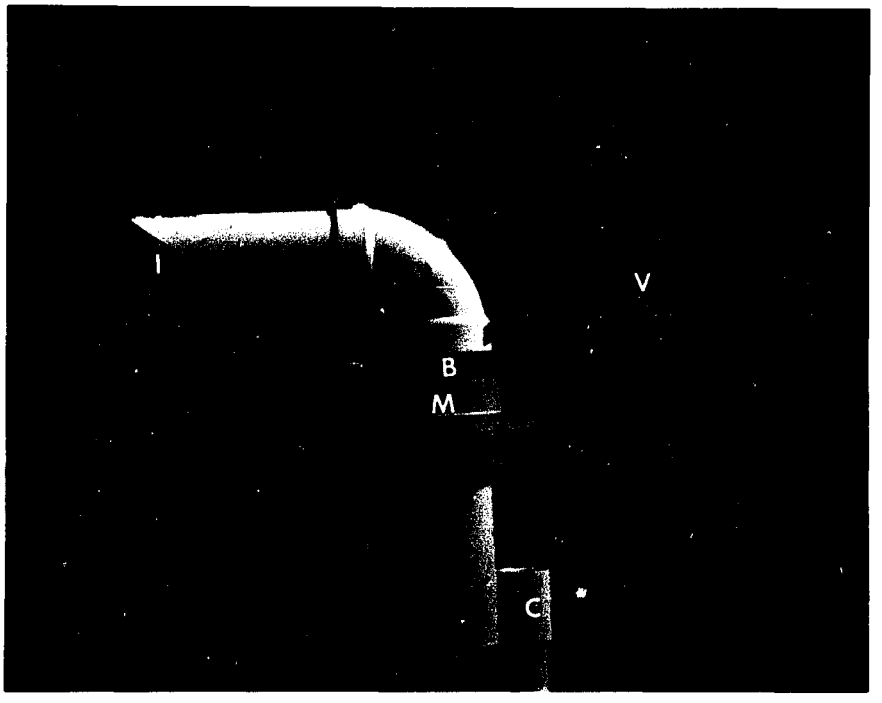



Figure 1. PVC spore trap used in field and glasshouse studies. V: windvane to point trap intake (I) into the wind. M: plastic molding housing the orifice and glass slide. B: reducing bushing in which bearing was placed. C: 1" coupler to mount trap onto 1" PVC tubing.

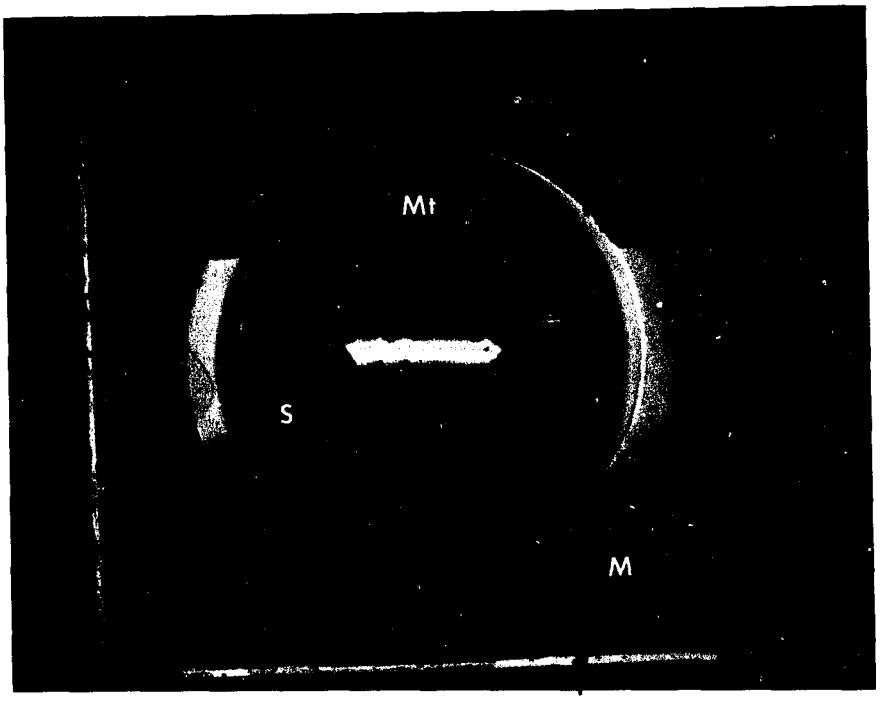



Figure 2. Glass slide (S) mounted in fitted depression in plastic molding (M). The white band represents a band of deposited spores corresponding to the trap orifice which is situated above the slide. Blower motor (Mt) is below the slide, out of focus.

# Appendix B

## Electric circuitry for operation of volumetric spore traps in the orchard

The electrical design for powering the volumetric spore traps in the orchard is presented in Figure 1. Current flowed from the 110 volt alternate currend (VAC) source to a variable transformer (Variac). The amount of current flowing from the variac to the battery charger could be controlled which, in turn, allowed control of the current flowing to the spore traps. This allowed operation of the spore traps over a range of sampling rates. The battery charger was used as a transformer and supplied direct current (DC) to power the spore traps.

The key to the system's operation was a solid state relay (Hamlin Model 7591 or equivalent) which allowed current from a 12-volt battery to throw a switch completing the 110 VAC circuit. Both the 110 VAC and the 12 VDC sources were connected to the solid state relay. A leaf wetness indicator (LWI) was connected to the 12 VDC battery and the relay. When the circuit was complete, current automatically flowed from the 110 VAC source to the spore traps, turning the traps on. When the string dried, the circuit from the LWI was broken and the traps were turned off. The LWI was also connected to a recording hygrothermograph, providing a record of hours of leaf wetness (and hence, spore trap operation).

Current flowed to each spore trap through 14 gauge wire (# vl202-

41-07, ITT Royal Electric, E. Providence, RI 02916). At each connection between a spore trap and the electrical wire, a 3/4 amp AGC fuse was attached as a safeguard so that if one trap failed, the rest of the system would remain in operation.

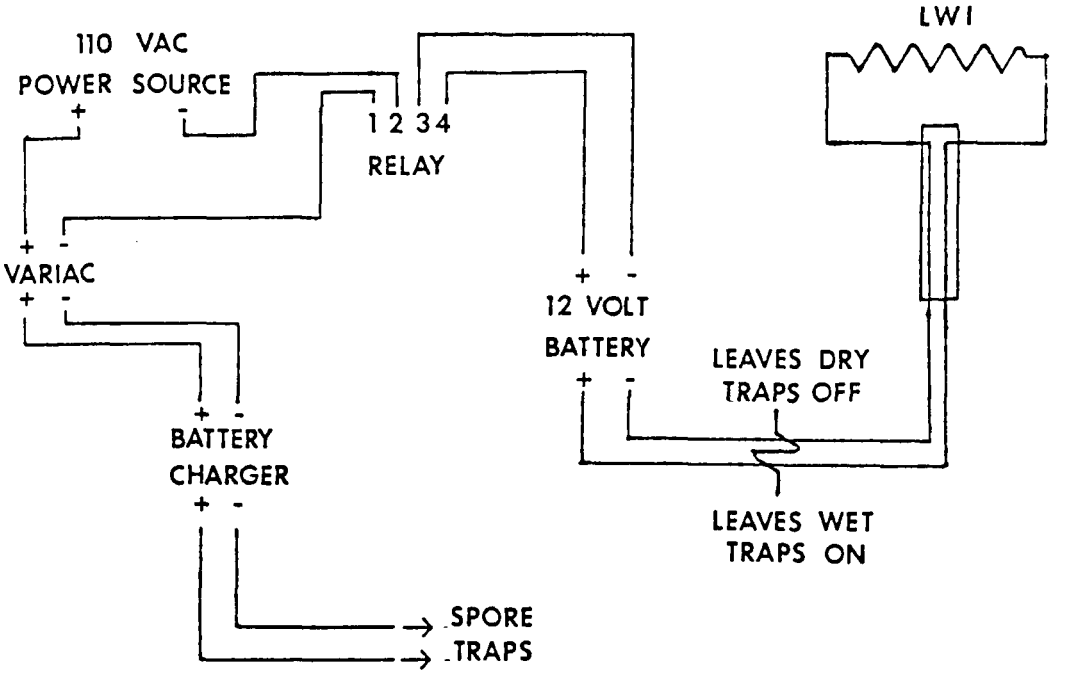



Figure 1. Electrical design for powering volumetric spore traps during orchard studies. VAC = volts, alternate current; VARIAC = variable transformer; LWI = Leaf Wetness Indicator.

# Appendix C

## Spore dispersal patterns for Lycopodium tests

This appendix contains results of dispersal tests using spores of Lycopodium sp which were presented in Chapter III. Figures 1-4 are the results of tests using design C (Fig. 3, Chapter III). In each figure, A, B, and C represent, respectively, the spore dispersal patterns in the first, second and third hour of each test. D is the pattern for the entire 3 hour period. Arrows represent the range in wind direction during each 3 hour test period.

Figures 5, 6 and 9 are the numbers of spores trapped in Lycopodium tests using designs B (Fig. 2, Chapter III), D (Fig. 5, Chapter III), and E (Fig. 6, Chapter III), respectively. In Figure 5, spore numbers are the total number of spores trapped at each site, which can be compared to the equivalent spore density used in the corresponding three dimensional graphs. Spore numbers in Figures 7 and 9 are the number of spores per $m^3$ air sampled.

Figures 6, 8, and 10 are the spore dispersal patterns for Lycopodium tests using designs B, D, and E, respectively. Where space has permitted, the spores trapped in each test and the spore dispersal patterns from the same tests have been placed on the same page.

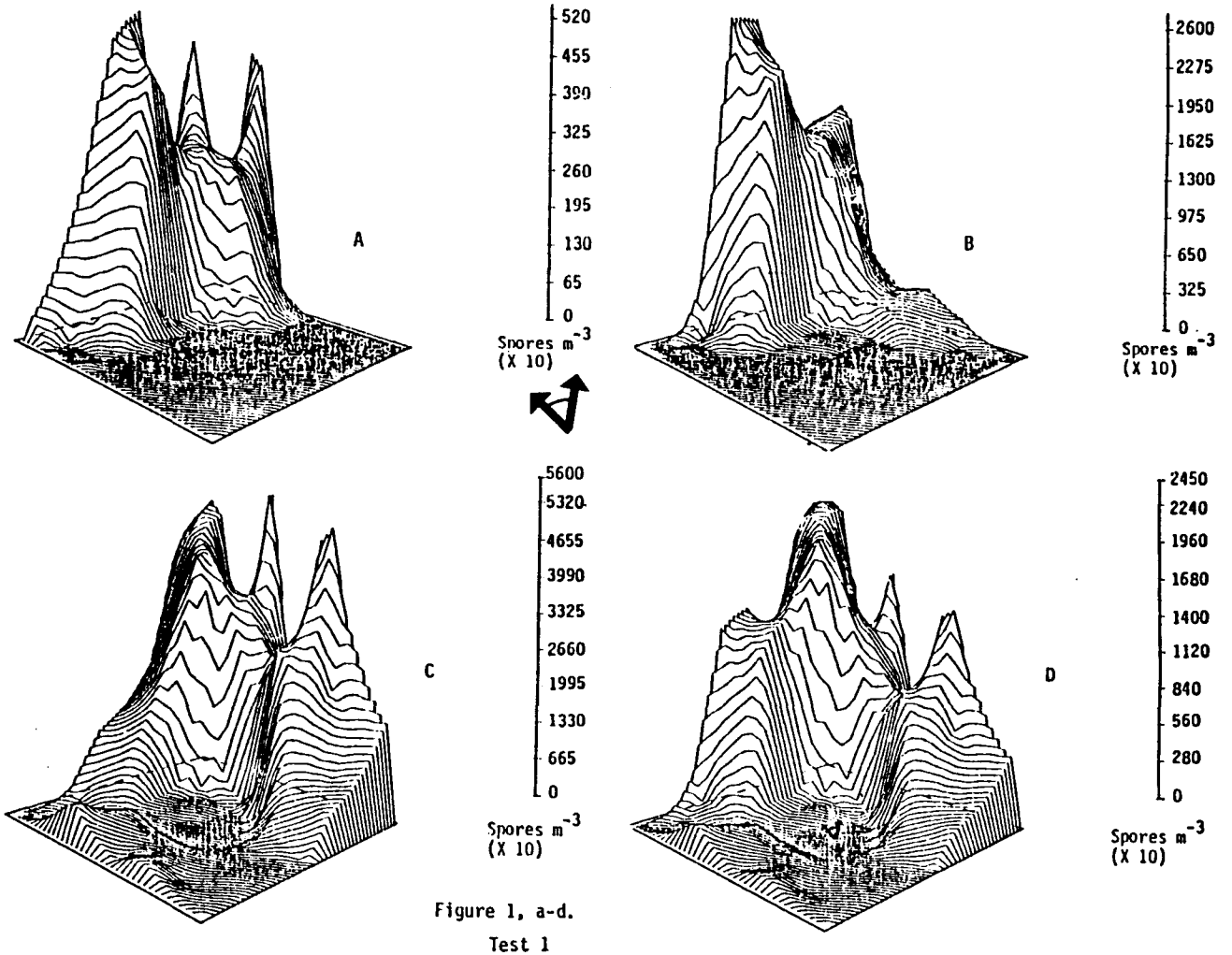



Figure 1, a-d.

Test 1

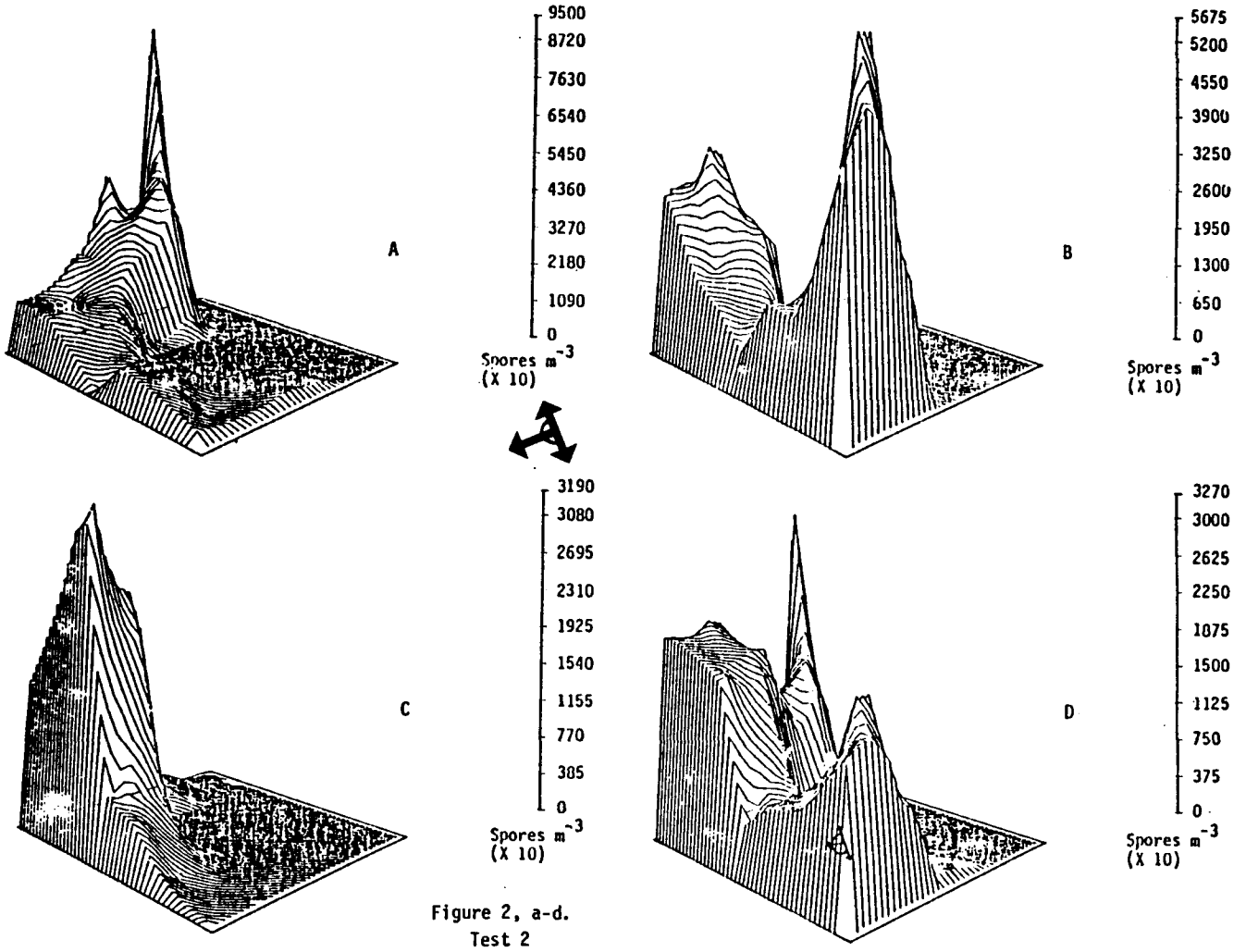



Figure 2, a-d.
Test 2

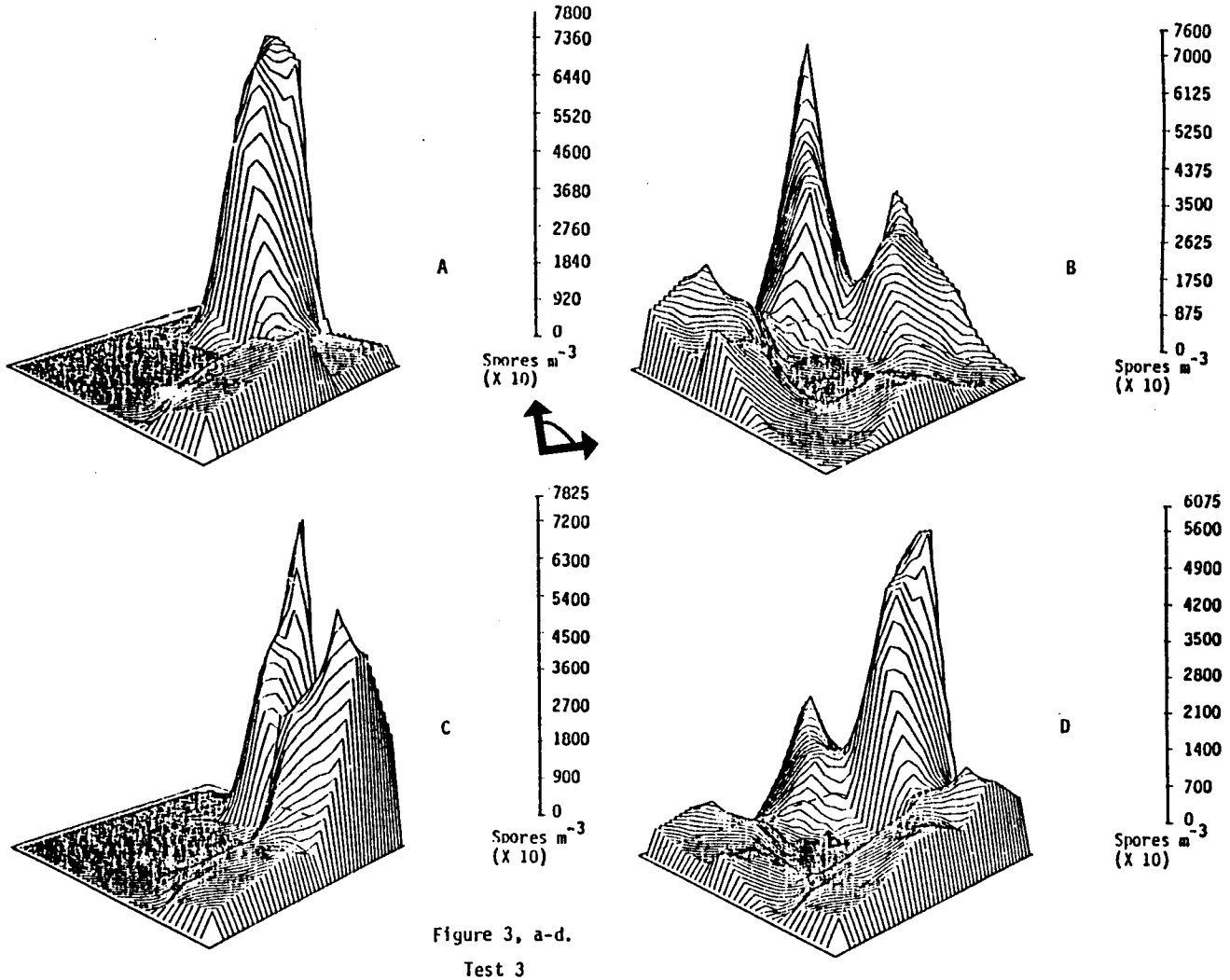



Figure 3, a-d.

Test 3

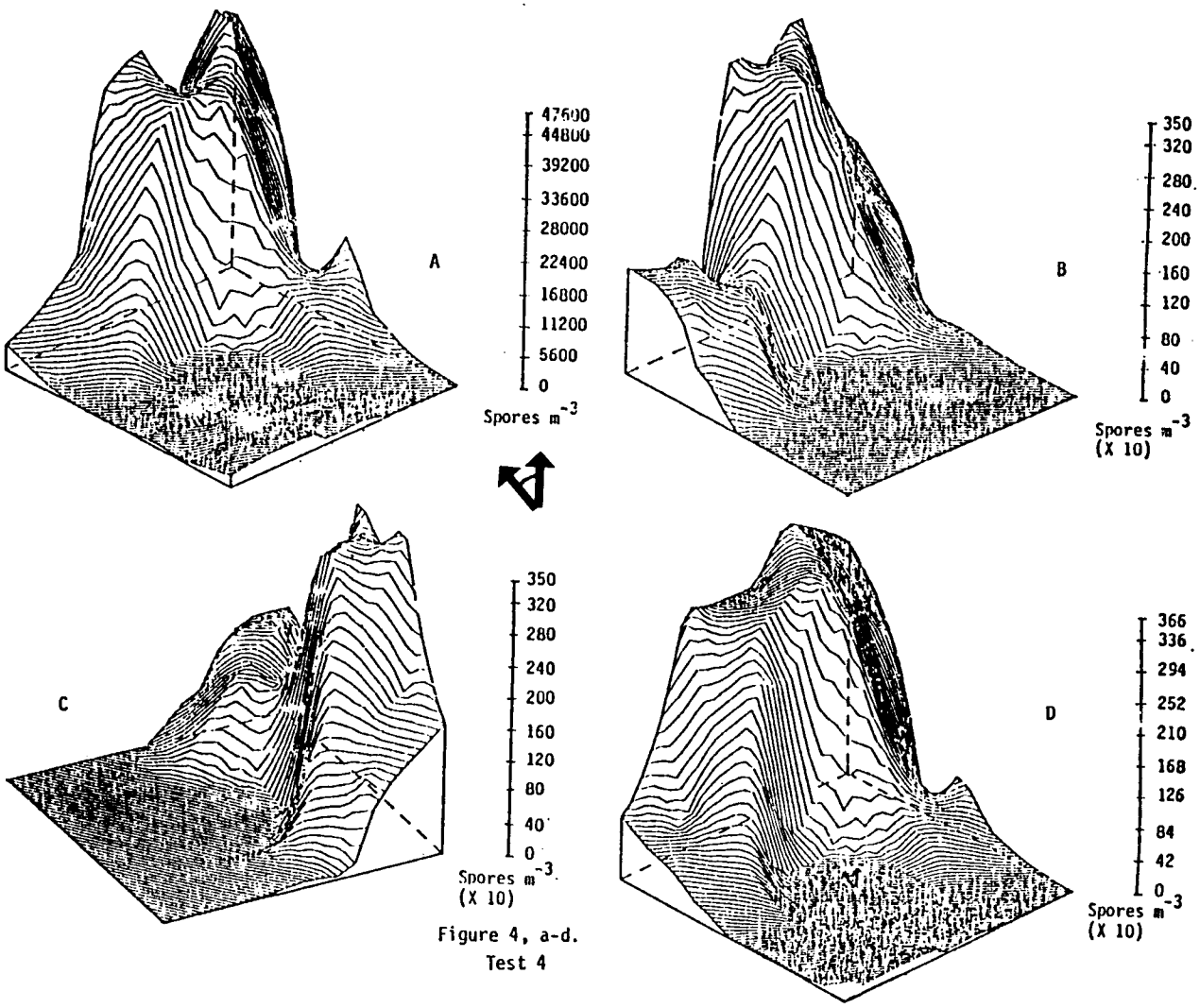



Figure 4, a-d.
Test 4

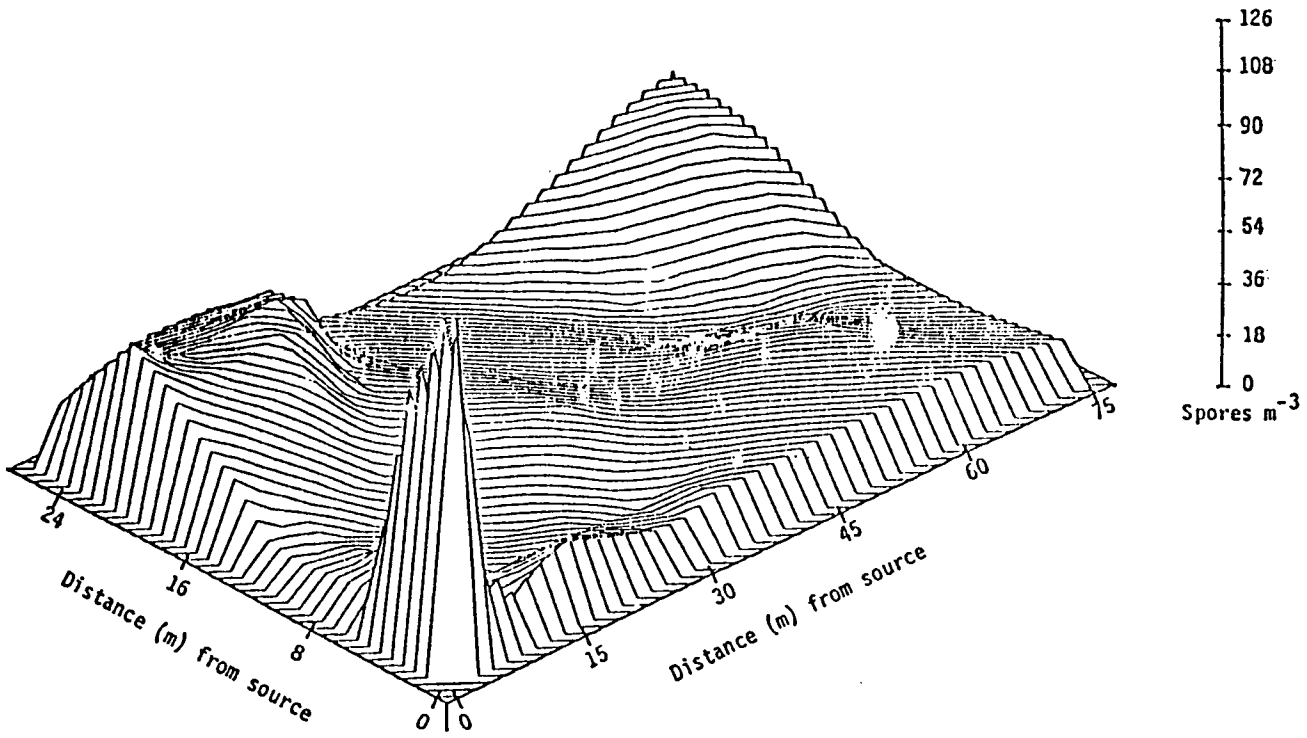



Figure 6a. Dispersal pattern, Test B1


Figure 5a. Spores trapped, Test B1

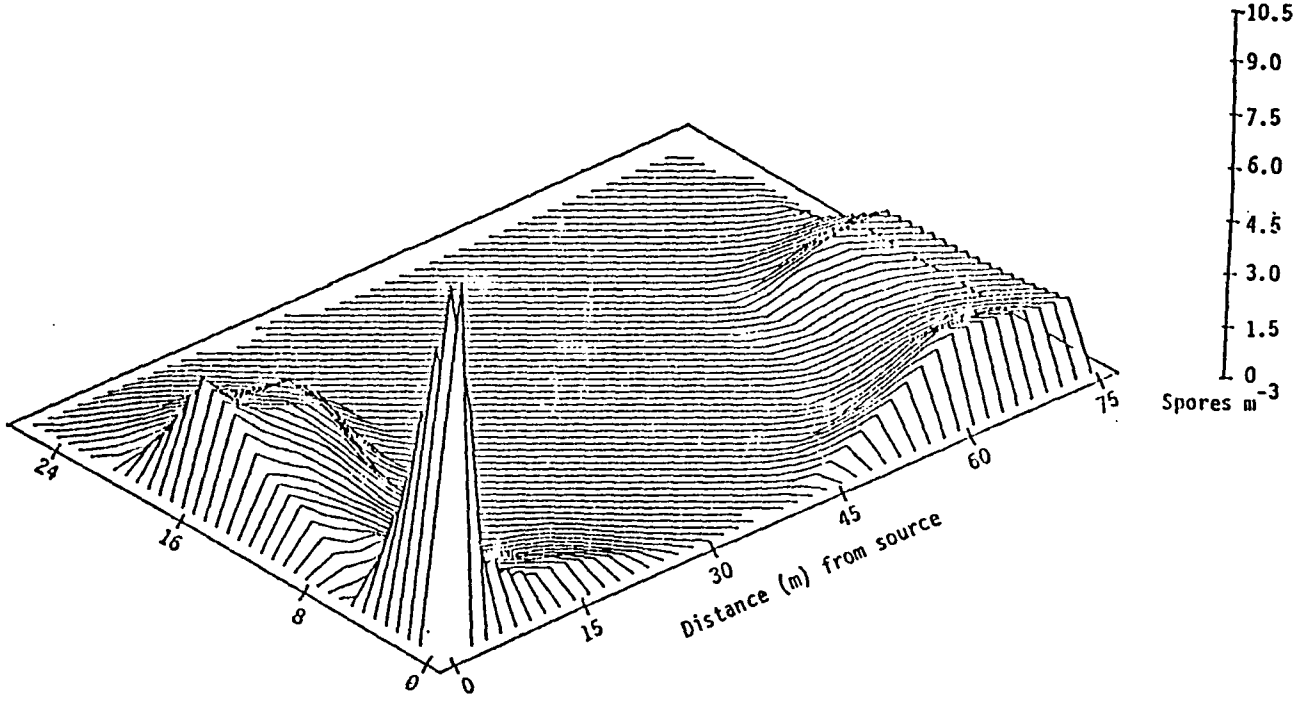



Figure 6b. . Dispersal pattern, Test B2


Figure 5b. Spores trapped, Test B2

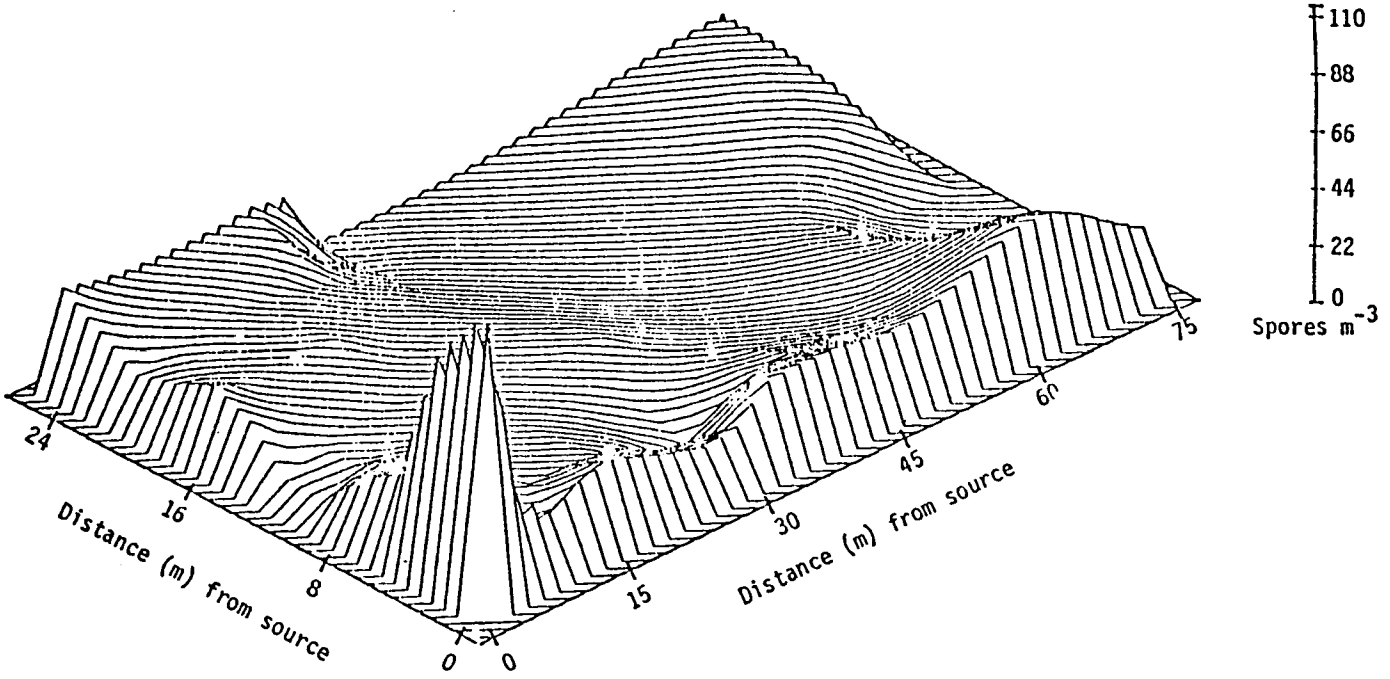



Figure 6c. Dispersal pattern, Test B3

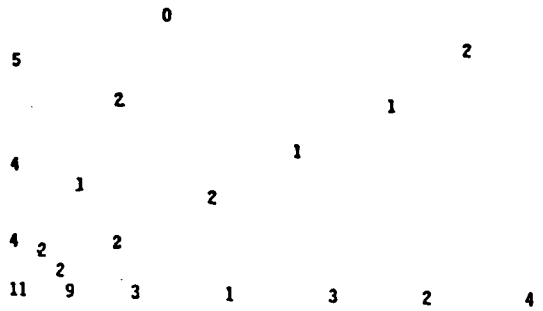



Figure 5c. Spores trapped, Test B3

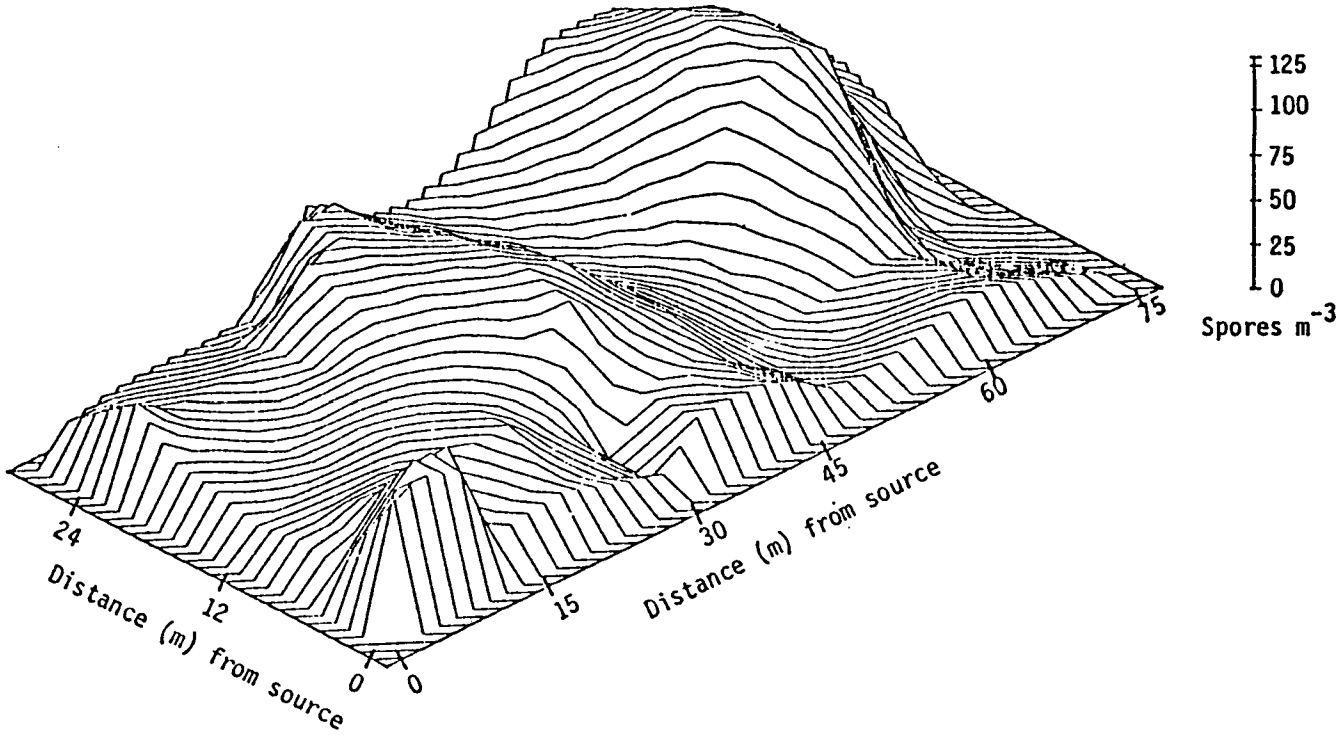



Figure 6d. Dispersal pattern, Test B4

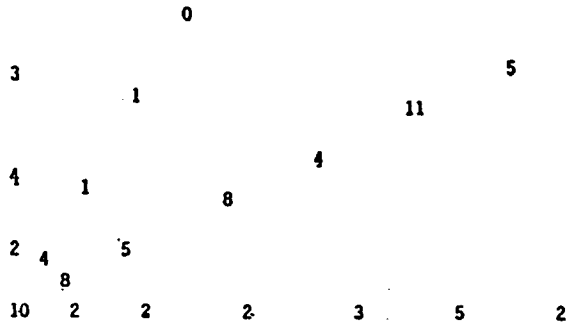



Figure 5d. Spores trapped, Test B4

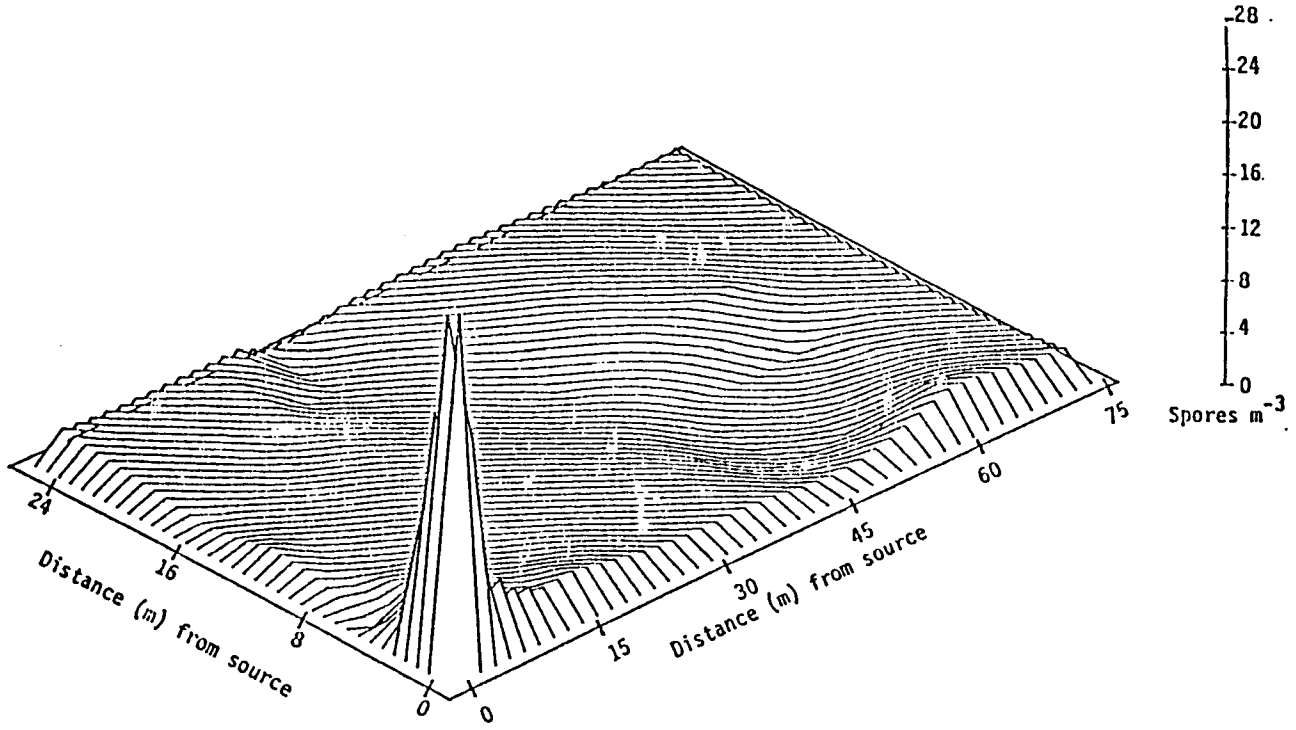



Figure 6e. Dispersal pattern, Test B5


Figure 5e. Spores trapped, Test B5

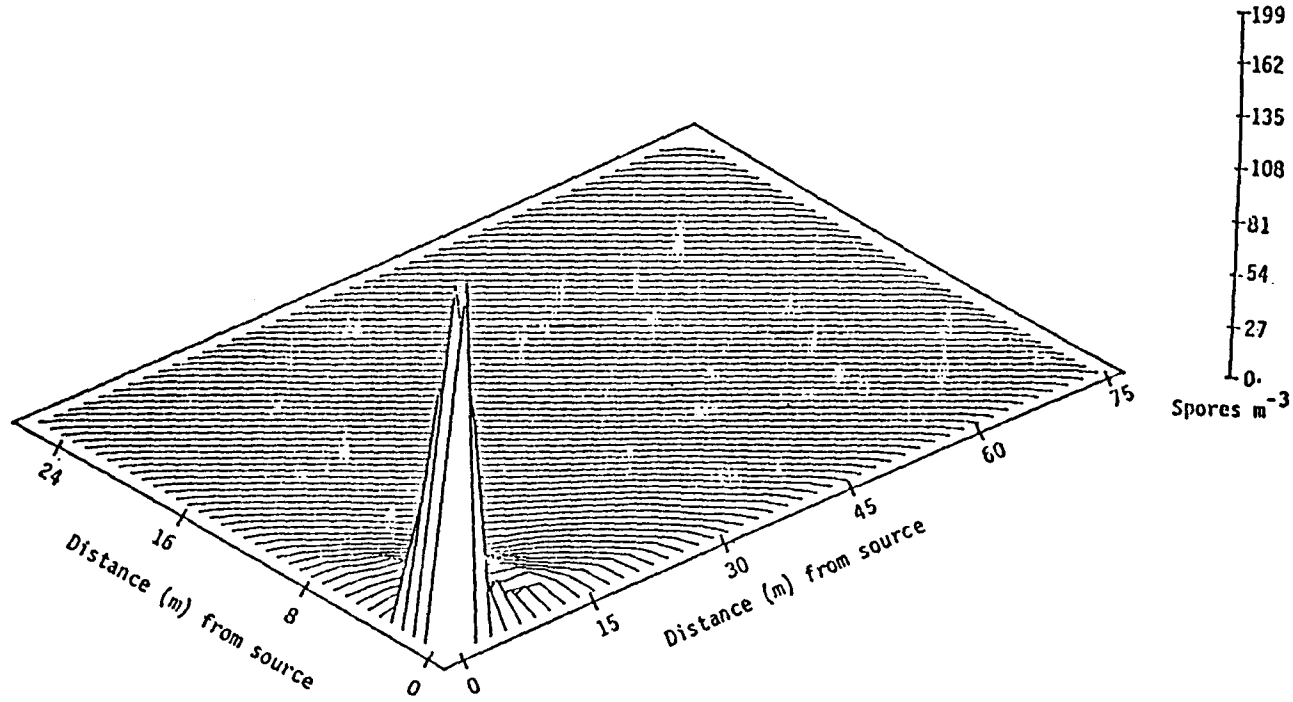



Figure 6f. Dispersal pattern, Test B6

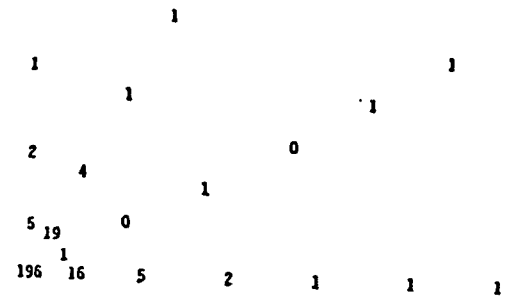



Figure 5f. Spores trapped, Test B6

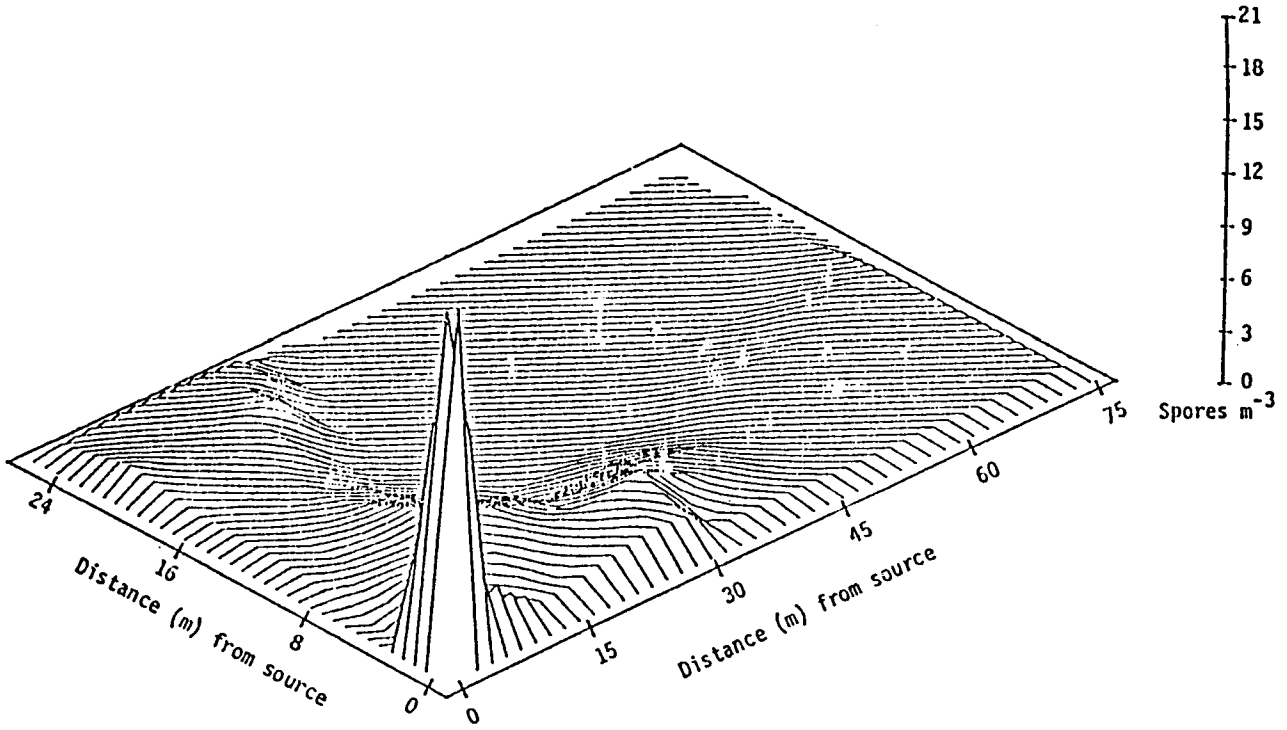



Figure 6g. Dispersal pattern, Test B7


Figure 5g. Spores trapped, Test B7

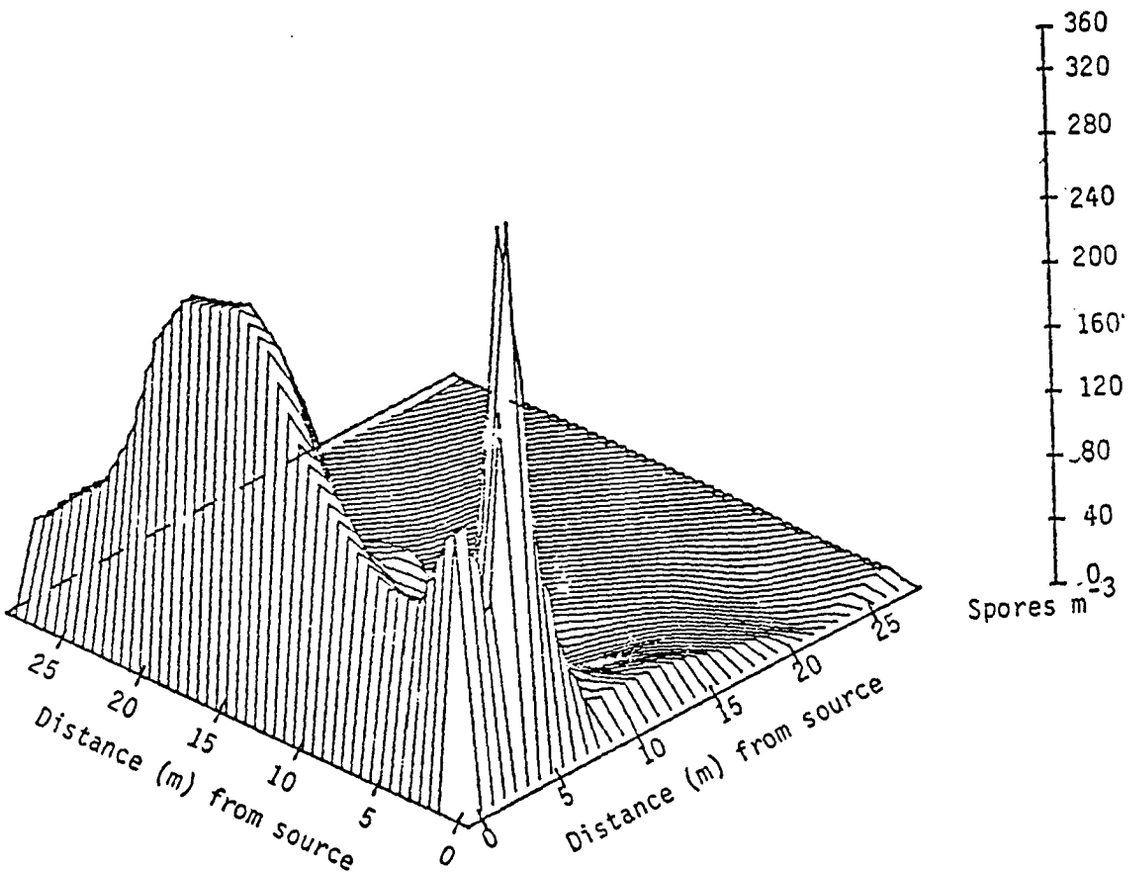



Figure 8a. Dispersal pattern, Test D1

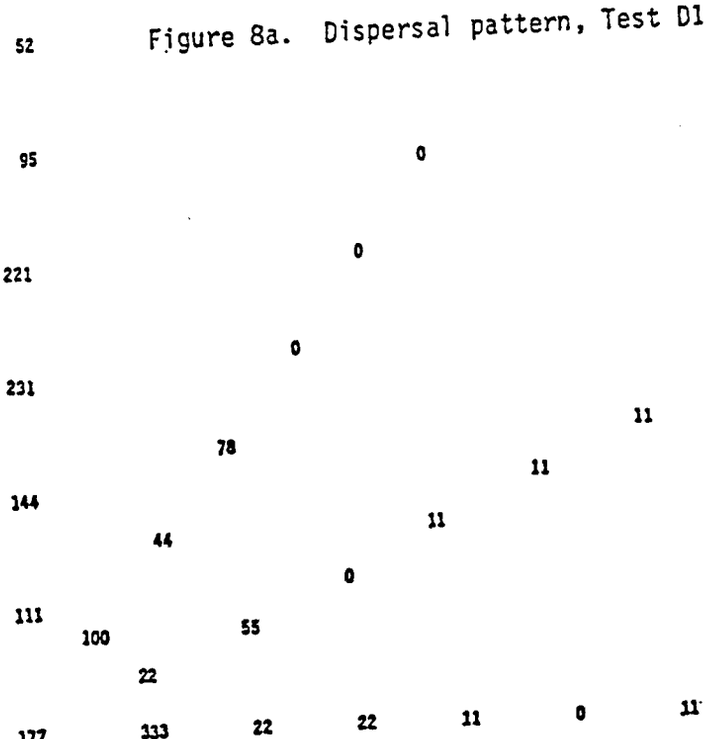



Figure 7a. Spores trapped, Test D1

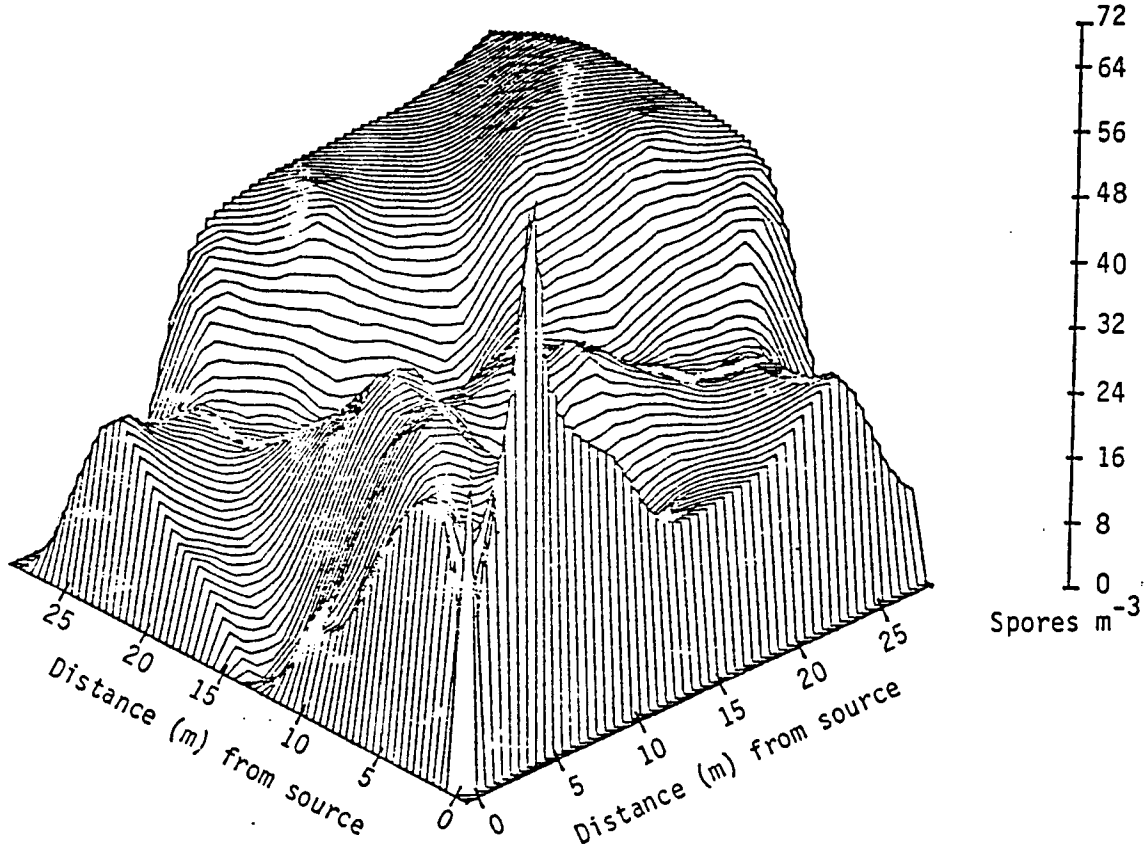



Figure 8b. Dispersal pattern, Test D2

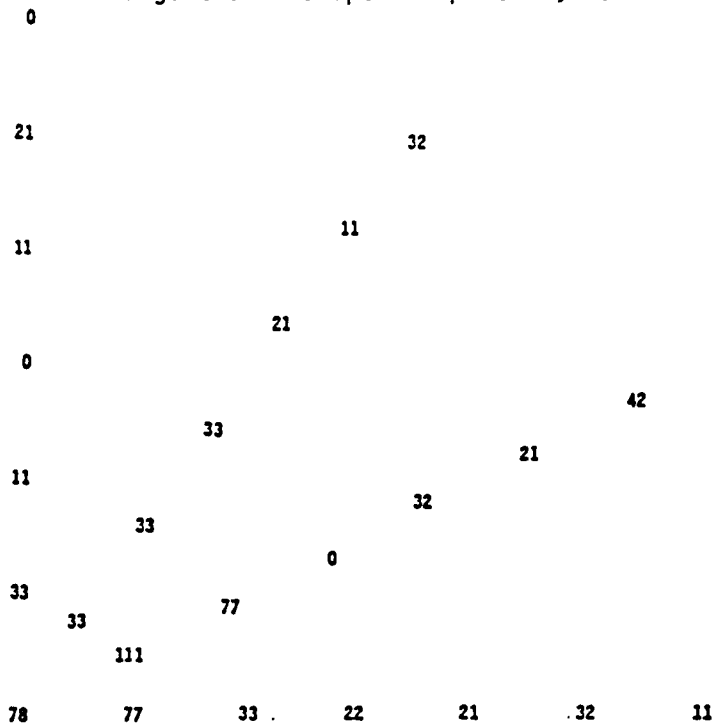



Figure 7b. Spores trapped, Test D2

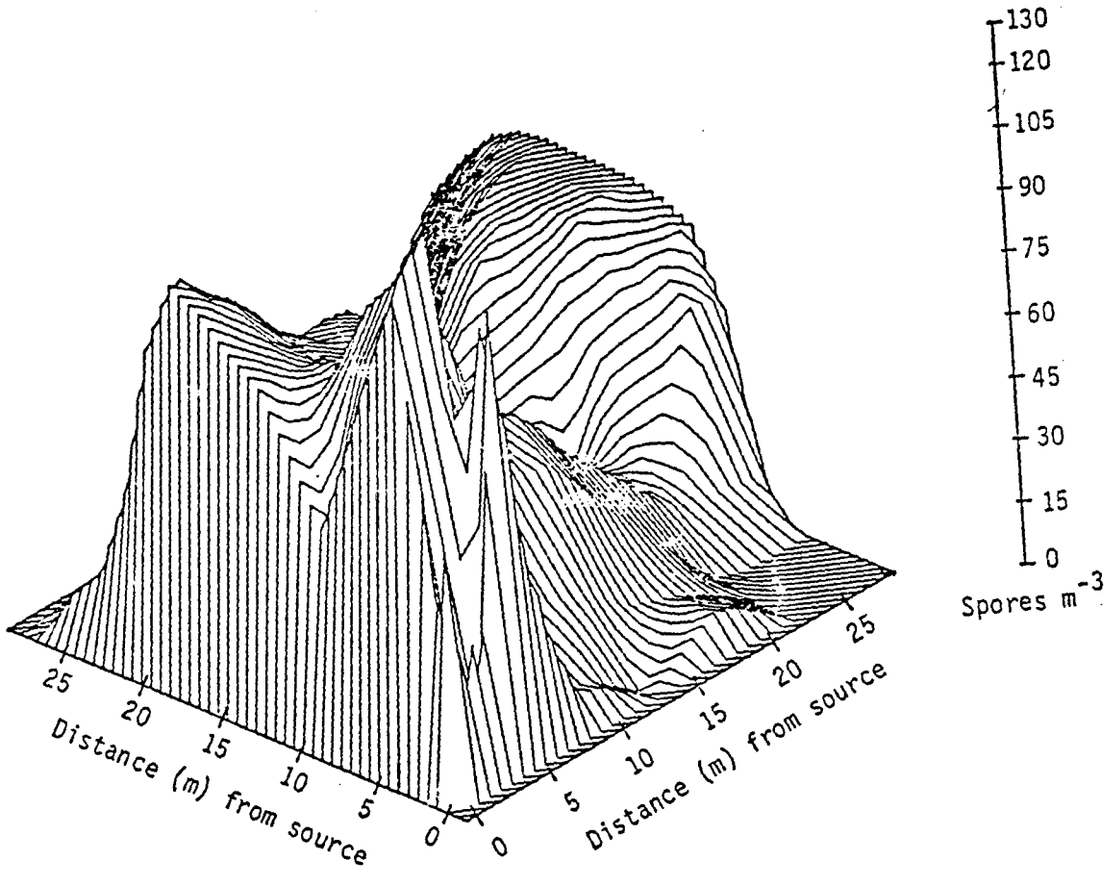



Figure 8c. Dispersal pattern, Test D3

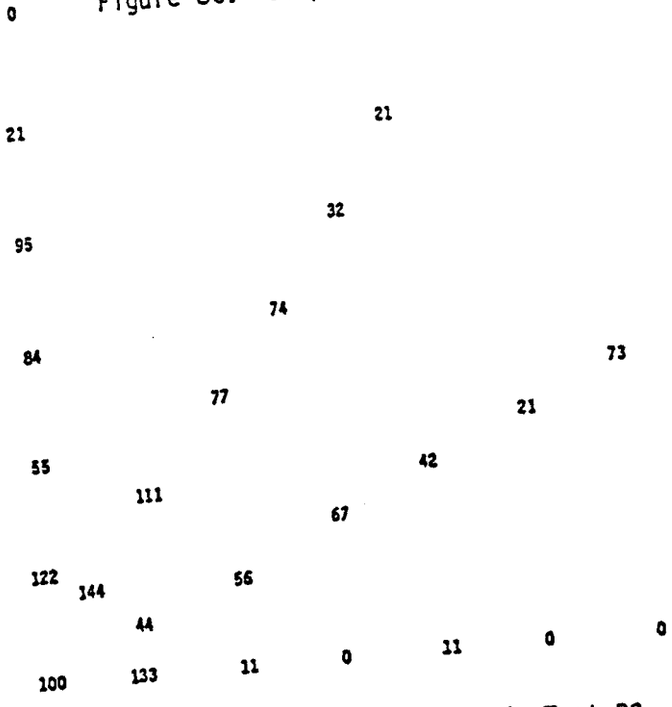



Figure 7c. Spores trapped, Test D3

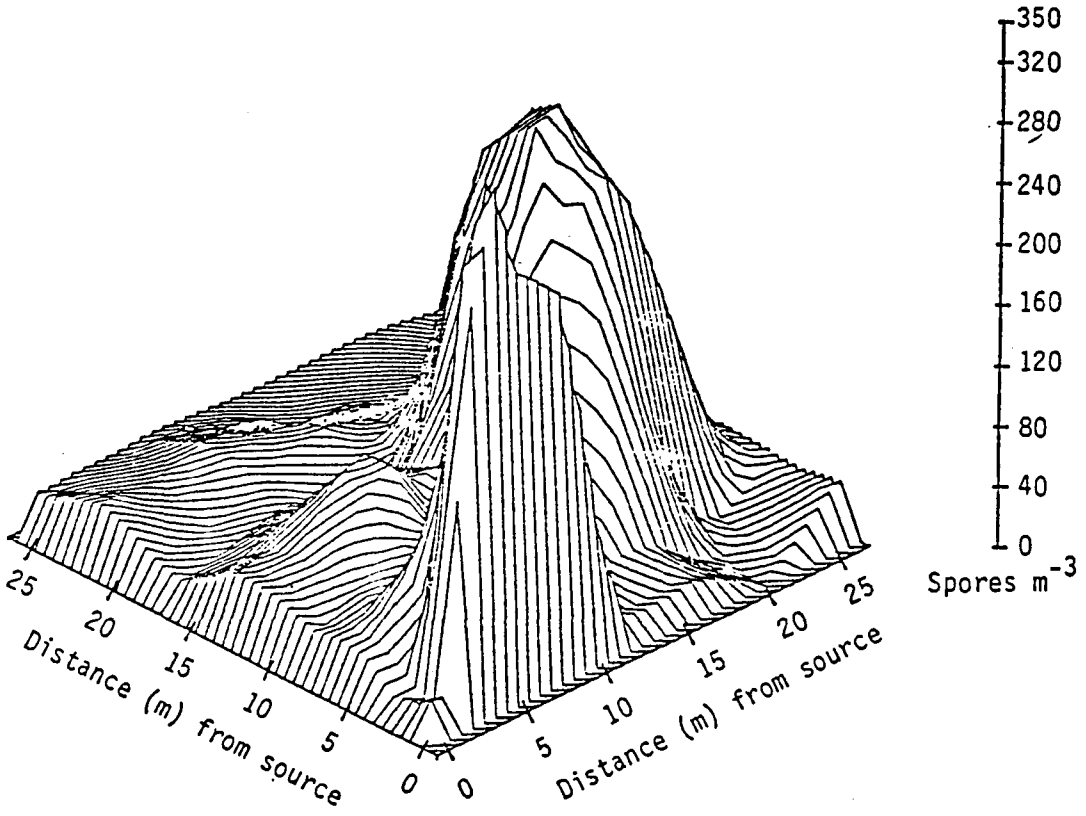



Figure 8d. Dispersal pattern, Test D4

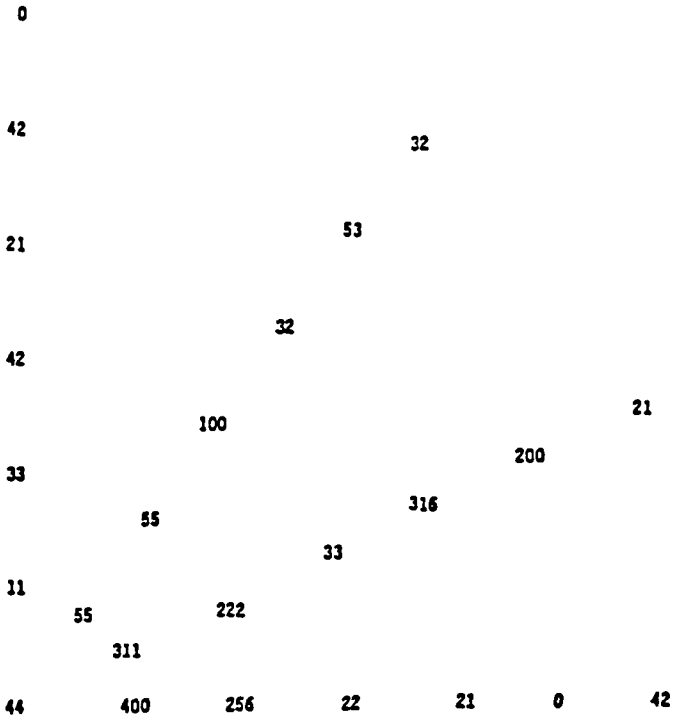



Figure 7d. Spores trapped, Test D4

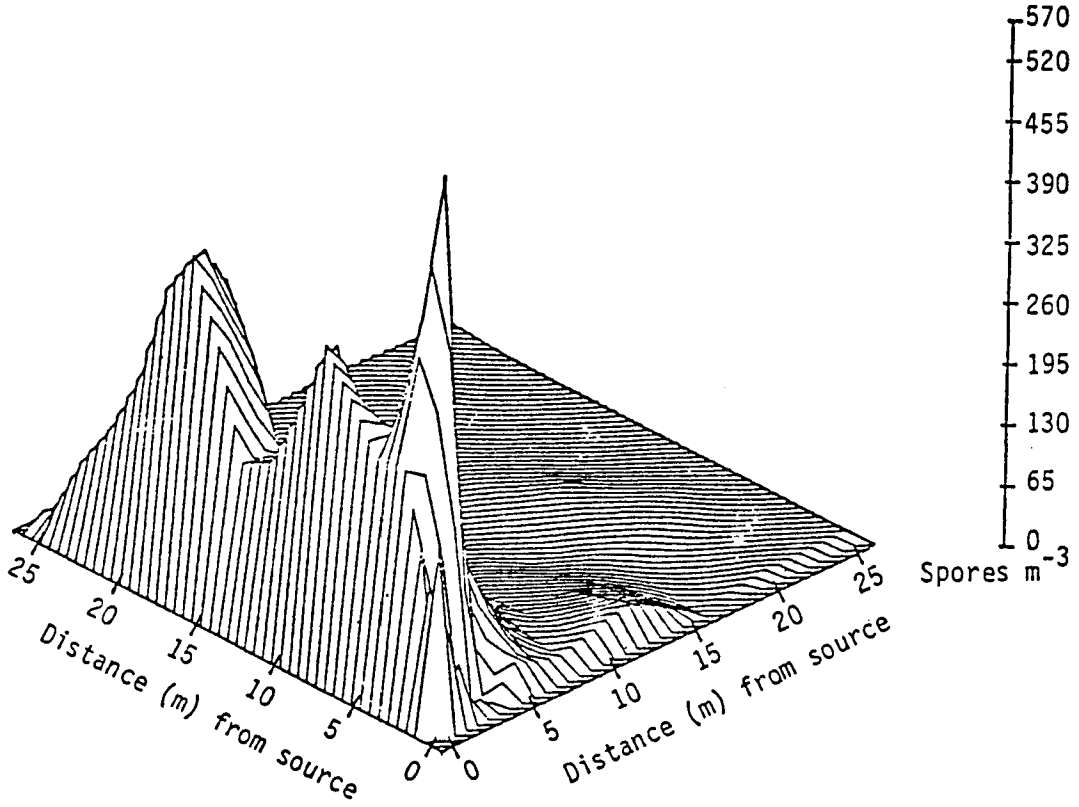



Figure 8e. Spore dispersal pattern, Test D5

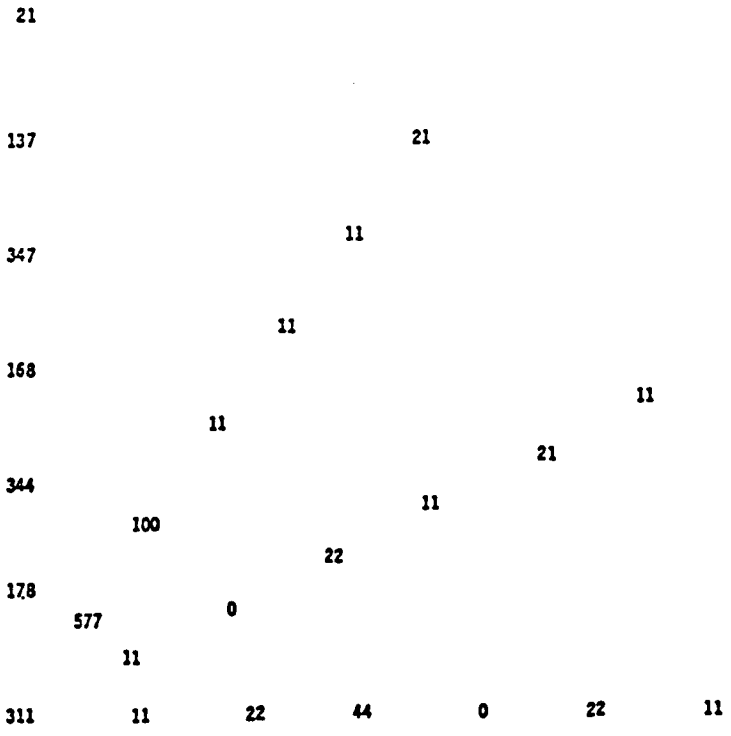



Figure 7e. Spores trapped, Test D5

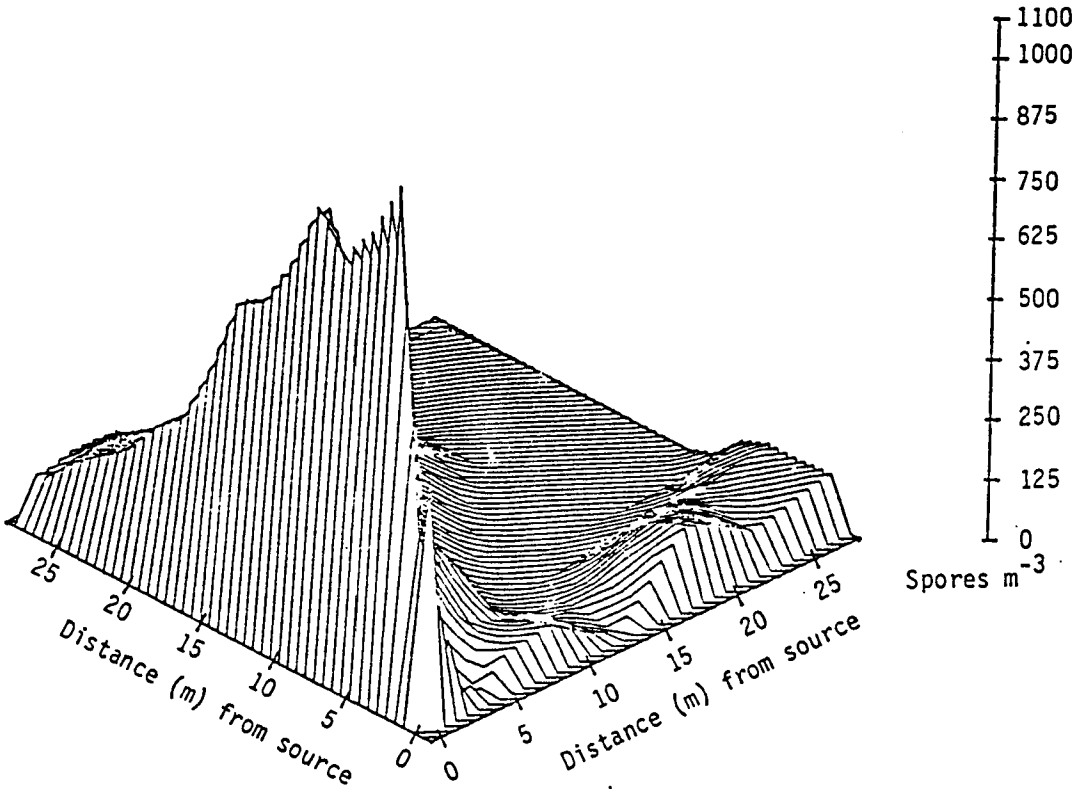



Figure 8f. Dispersal pattern, Tesc D6

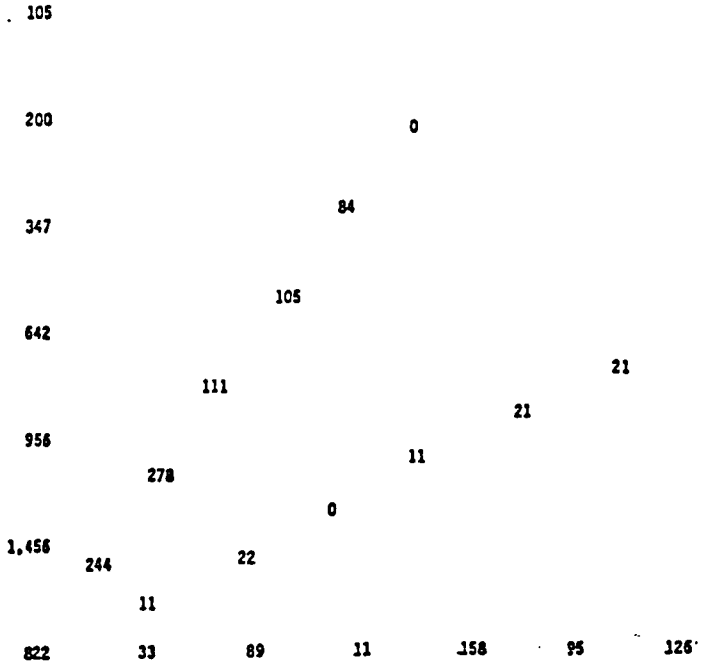



Figure 7f. Spores trapped, Test D6

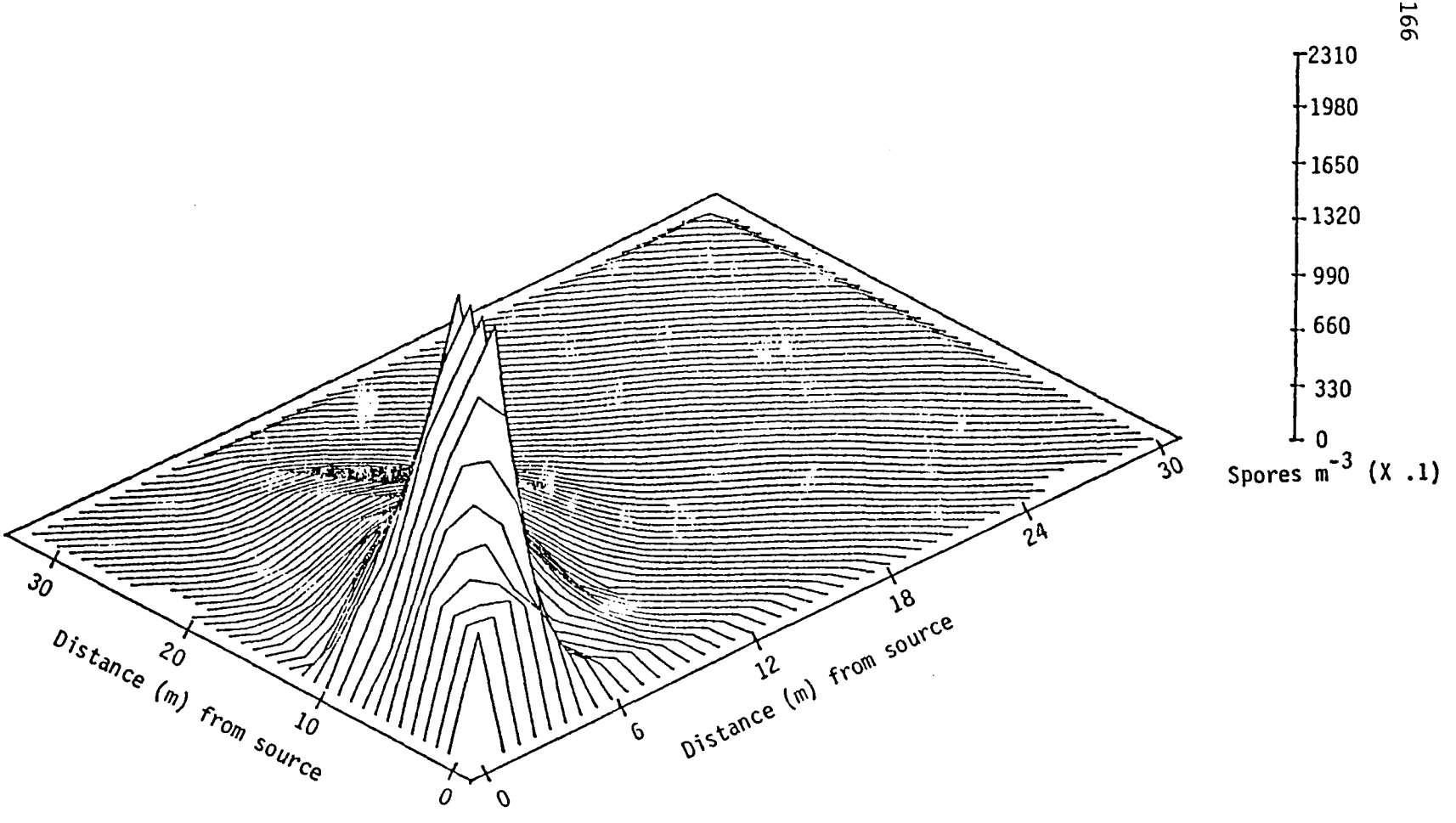



Figure 9a. Spore dispersal pattern, Design E, Test 1.

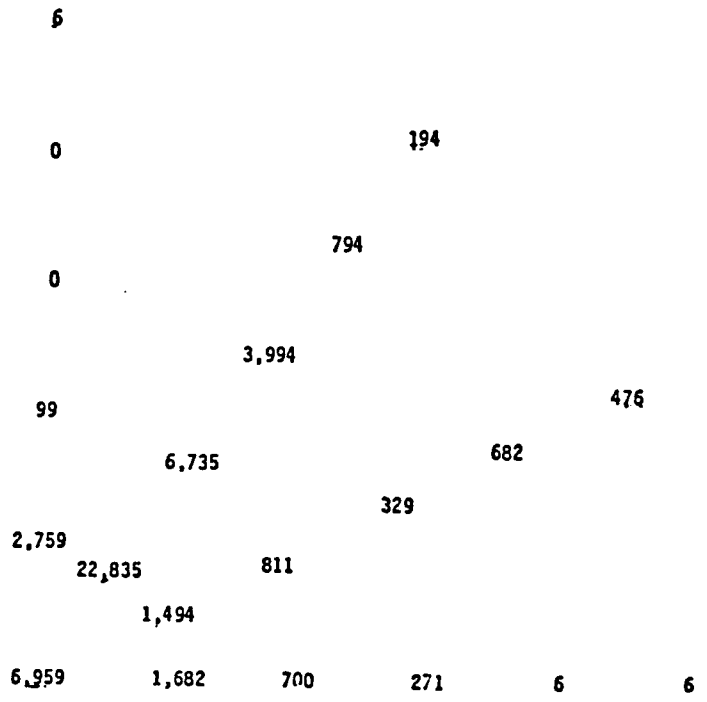



Figure 10a. Spores trapped ($m^{-3}$ air), design E, test 1.

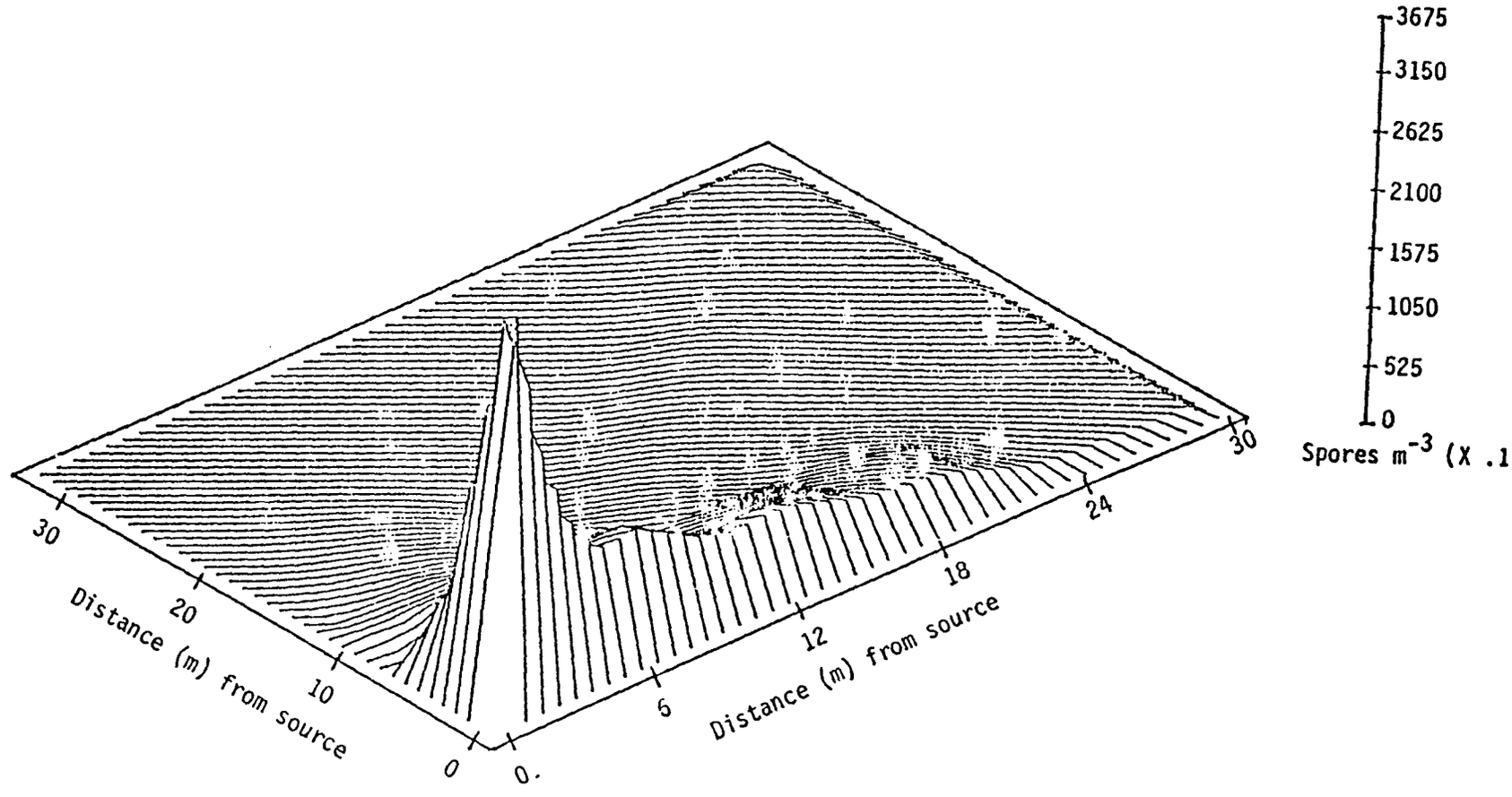



Figure 9b. Spore dispersal pattern, Design E, Test 2.

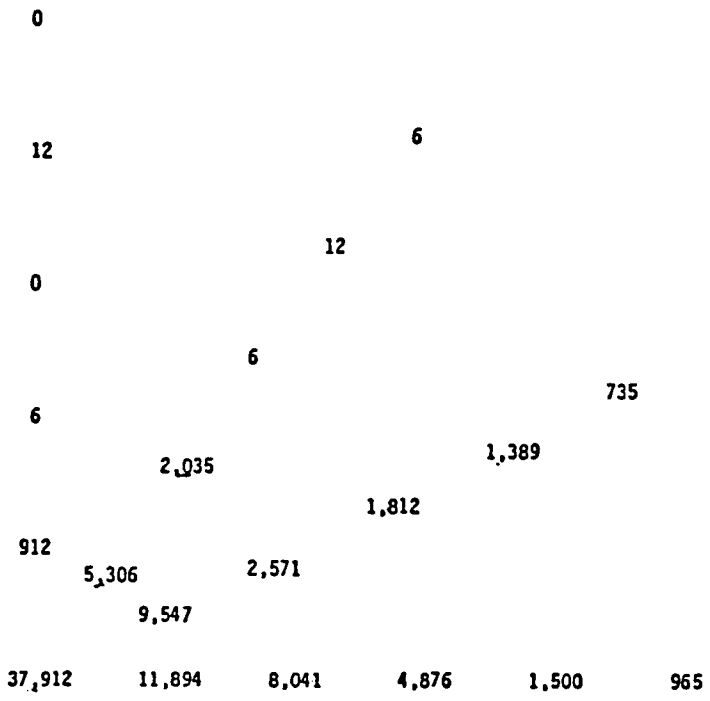



Figure 10b. Spores trapped ($m^{-3}$ air), design E, test 2.

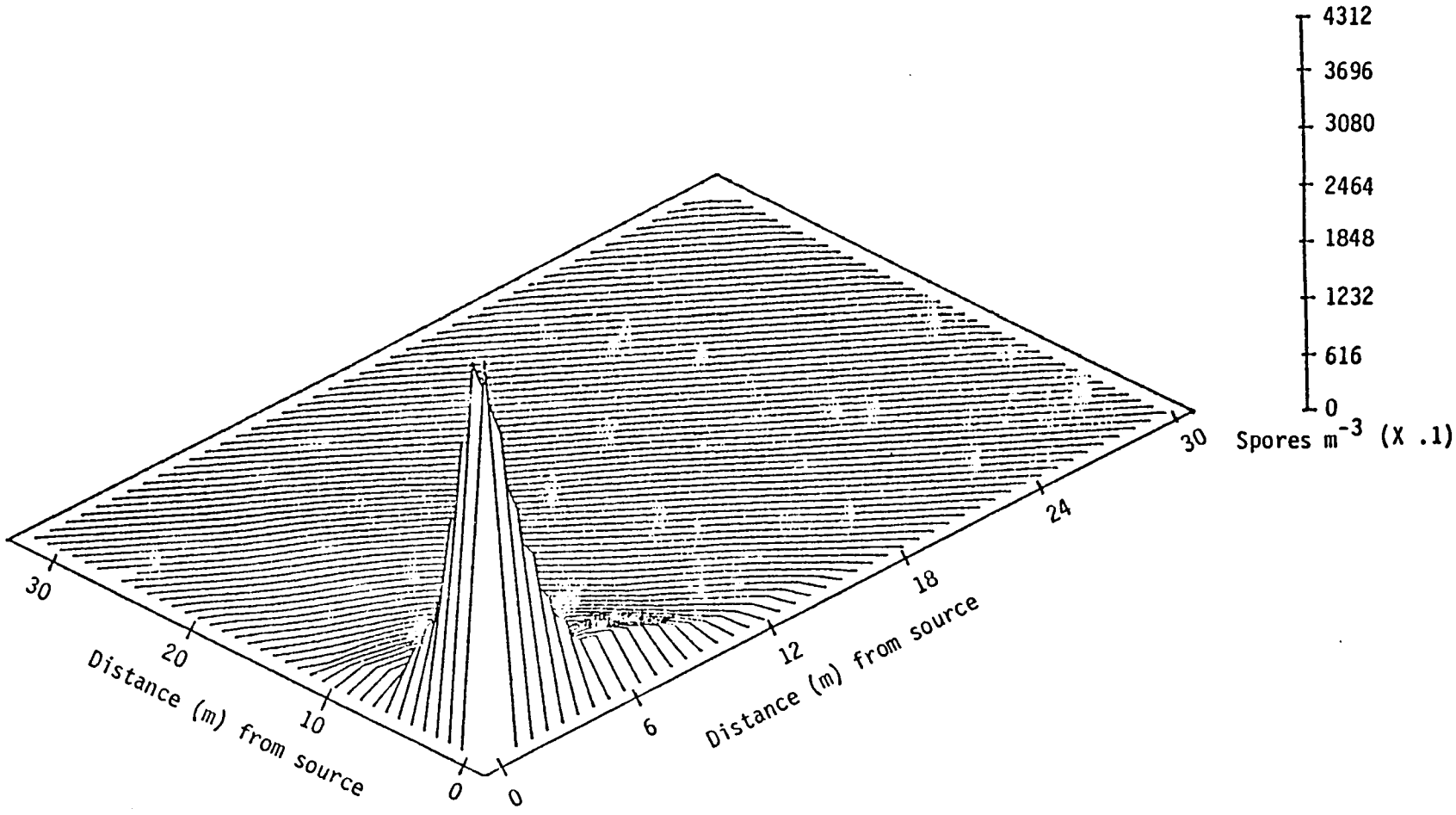



Figure 9c. Spore dispersal pattern, Design E, Test 3.

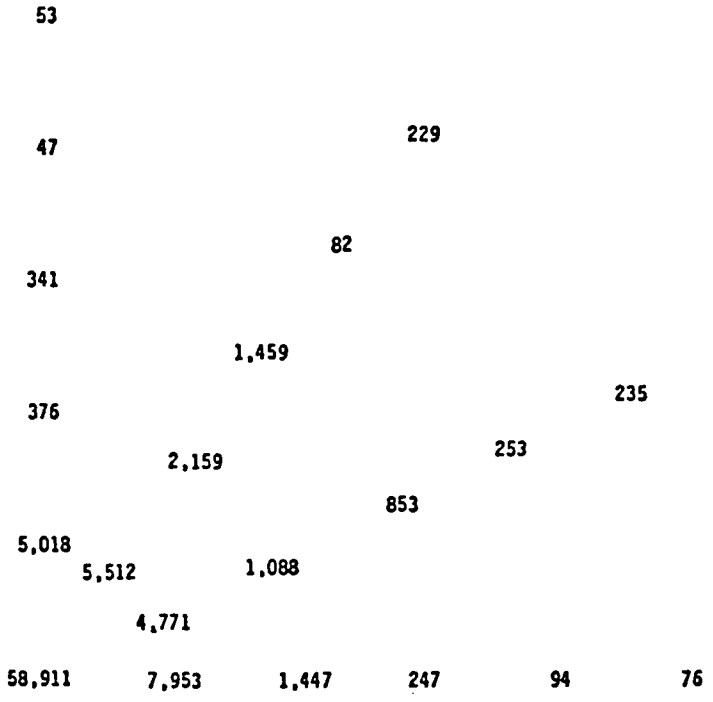



Figure 10c. Spores trapped ($m^{-3}$ air), design E, test 3.

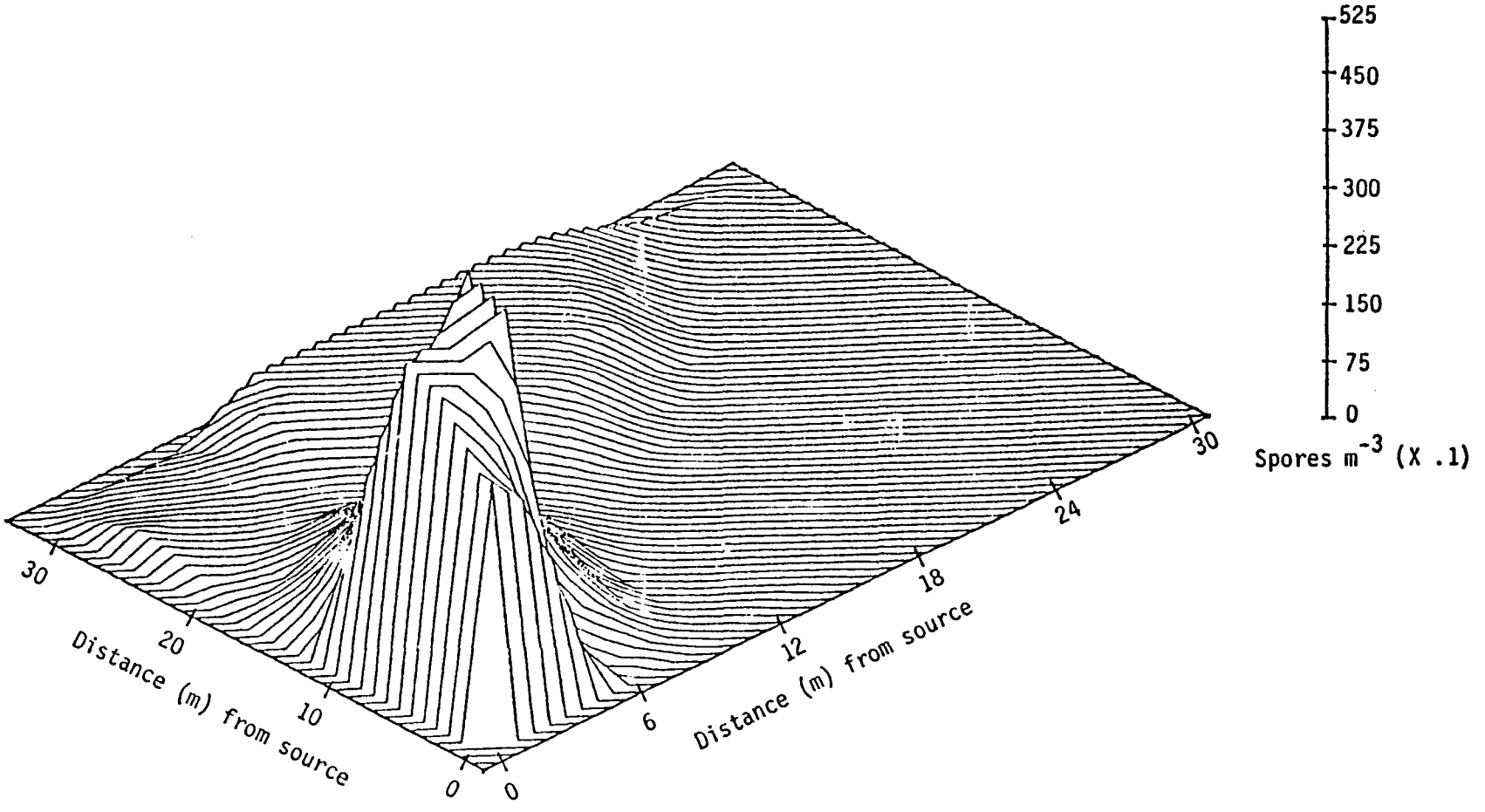



Figure 9d. Spore dispersal pattern, Design E, Test 4.

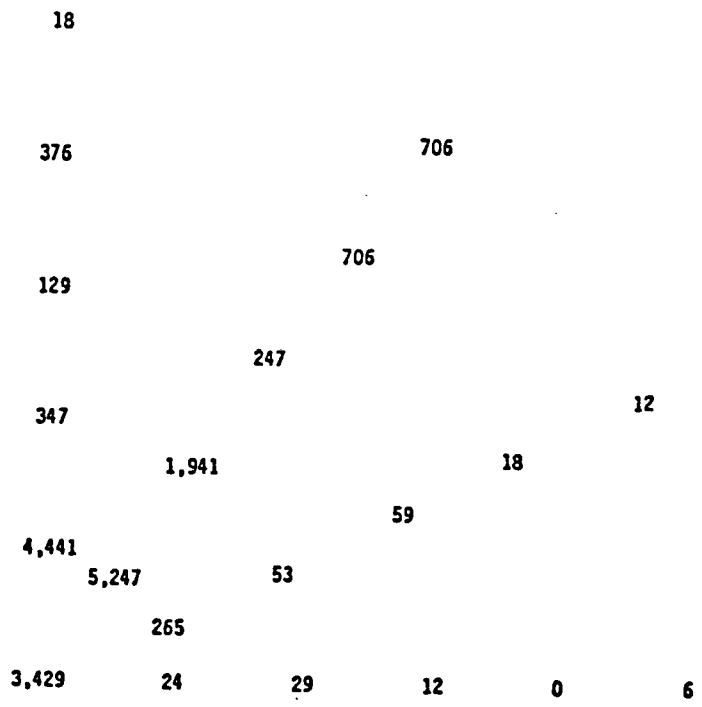



Figure 10d. Spores trapped ($m^{-3}$ air), design E, test 4.

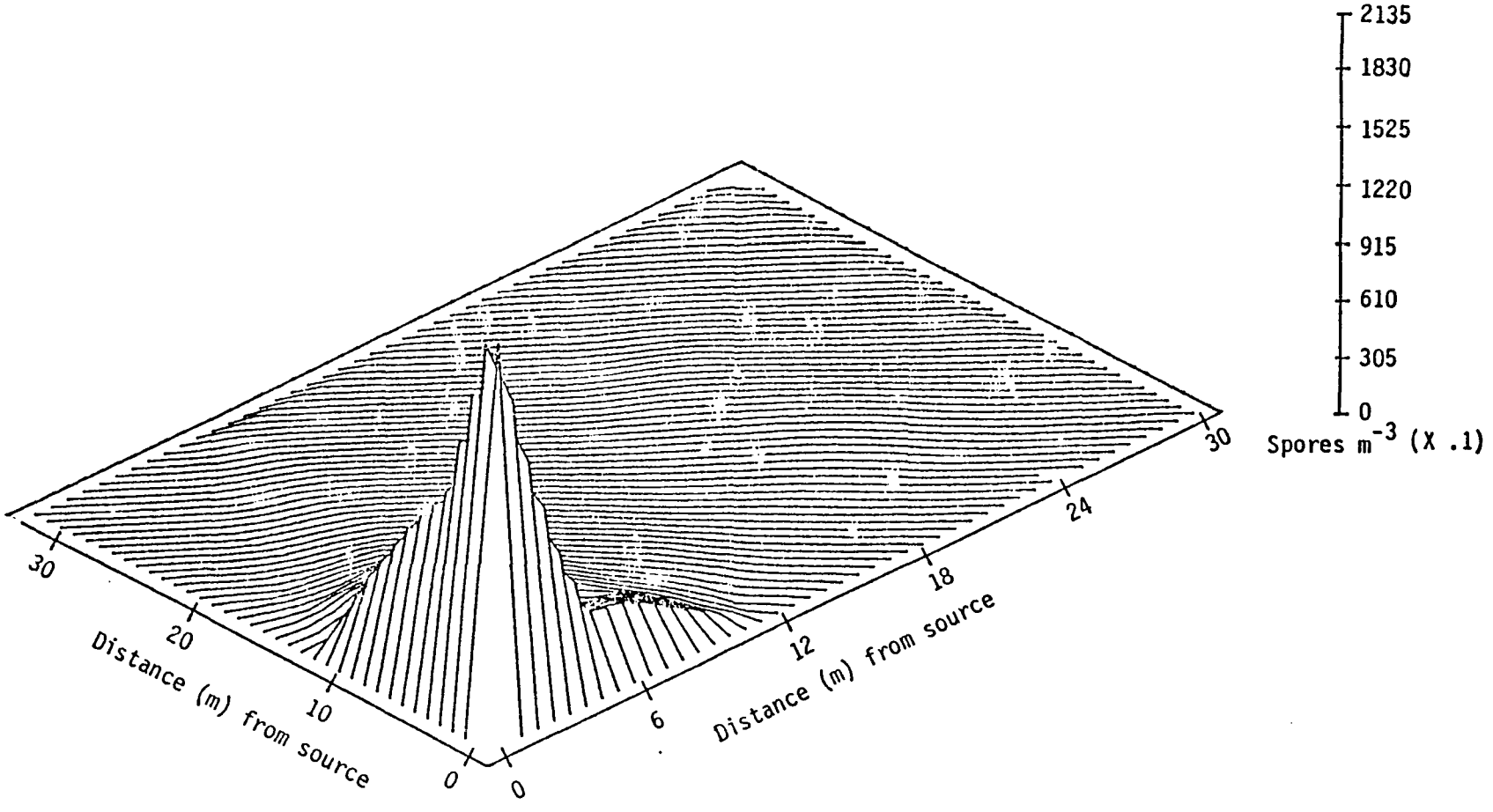



Figure 9e. Spore dispersal pattern, Design E, Test 5.

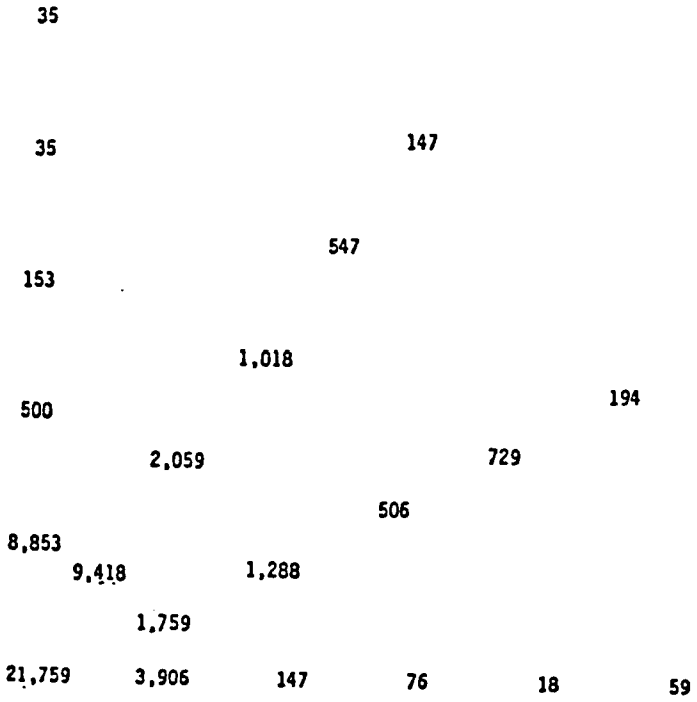



Figure 10e. Spores trapped ($m^{-3}$ air, design E, test 5.

# Appendix D

## Summary of meteorological conditions during 1984 and 1985 primary infection periods.

The infection periods described in this appendix occurred during the 1984 and 1985 primary scab seasons. For each infection period described, one plot of trees at the Woodman Horticulture Farm was unprotected with fungicide. For ease of reference the plots unprotected for each infection period, as described in Chapter IV, are presented with each meteorological summary. All records of time of day are Eastern Standard Time (EST)

### 1984

**Infection period, 3-5 May (Plot A).** Heavy rain fell from 2000 hours on 3 May until 0900 hours on 4 May. Light, intermittent showers continued until 2100 hours. Leaves were dry by 1400 hours on 5 May after 41 hr of continuous leaf wetness. The average temperature during the interval was 4.5 C. Wind speeds were 2.3-9 m $s^{-1}$, from E to NE until 1600 hours on 4 May, then shifting to N and NW.

**Infection period, 8-9 May (Plot B).** Heavy rain began at 1200 hours on 8 May and continued until 1900 hours that evening. Leaves were dry by 0700 hours on 9 May after 19 hr of continuous leaf wetness. The average temperature during the interval was 7 C. The predominant wind

direction was from the E during the afternoon of 8 May, with speeds of 0.9-6.7 m $s^{-1}$. After 1900 hours, the wind shifted towards the NE and diminished to 0-3 m $s^{-1}$ for the remainder of the infection period.

**Infection period, 12-13 May (Plot C).** A light, steady, mist-like rain began at 0200 hours on 12 May and continued until 2100 hours the same day. Leaves were dry by 0500 hours on 13 May after 27 hr of continuous leaf wetness. The average temperature during the interval was 11.5 C. Winds were light and variable with an average speed of 0-3 m $s^{-1}$.

Moderate, steady rain began again at 2100 hours on 13 May and continued until 0900 hours on 14 May. At 1300 hours on 14 May, the wind shifted from NE to NW and the relative humidity dropped from 95% to 50% in 90 minutes, the result being that the leaves were dry by 1400 hours on 14 May, after 17 hr of continuous leaf wetness. The average temperature during the interval was 8 C. According to Mills (90), this second period of leaf wetness would be considered an infection period. However, based on evidence from a previous study (78) and the spore trapping data from this study, few ascospores were released until 0900 hours on 14 May. The 5 hr of leaf wetness (0900-1400 hours) during spore release was not sufficient (77) to consider the second rainy interval an infection period.

**Infection period, 28 May-2 June (Plot D).** Heavy rain began at 1030 hours on 28 May and continued until 1600 hours on 1 June. By 1600 hours on 28 May, 9.5 cm of rain had fallen, and an additional 4.4 cm

fell during the next 48 hr. Leaves were dry by 0700 hours on 3 June, after 140 hours of continuous leaf wetness. The average temperature during this interval was 13 C. Wind speed was light, with an average speed of 0-4.5 m $s^{-1}$ throughout the entire infection period. Wind direction was variable, predominantly from the NE, but also from the SE during the first 54 hr of the infection period.

## 1985

**Infection period, 5-7 May (Plot A).** Moderate rain began at 1430 hours on 5 May and continued until 1700 hours on 6 May. Leaves were dry by 0700 hours on 7 May, after 39.5 hr of continuous leaf wetness. The average temperature during the interval was 6.1 C. Wind direction throughout the infection period was NE to E, steady (2-6 m $s^{-1}$) with gusts to 8.9 m $s^{-1}$.

**Infection period, 17-19 May (Plot B).** Light rain began at 2300 hours on 17 May and continued until 1000 hours on 18 May. At 1400 hours there was a heavy downpour. The sky was clear by 1600 hours, but the leaves remained wet until 2200 hours that night. A brief shower at 0200 hours on 19 May rewet the leaves, which did not dry until 1000 hours on 19 May. The duration of the infection period, including the 4 hr on 18 May when the leaves were dry was 35 hours. The average temperature during the infection period was 7.7 C. Winds throughout the period were initially light (0-4 m $s^{-1}$) from the SE, E and NE but became Westerly and strong (steady to 11.1 m $s^{-1}$) at 1400 hours on 18

May. Winds diminished by 0000 hours on 19 May and remained light and variable throughout the remainder of the infection period.

**Infection period, 21-22 May (Plot C).** A thunderstorm occurred at 1330 @hours, followed by showers that continued until 2200 hours. The leaves were dry by 0600 hours on 22 May after 16.5 hr of continuous leaf wetness. The average temperature during the interval was 13.9 C. Winds during the period were calm to very light (0-8.9 m $s^{-1}$), variable or from the SE when measurable.

**Infection period, 27-29 May (Plot D).** Light showers began at 1530 hours on 27 May, and continued intermittently until 1700 hours on 28 May. Leaves were dry by 0600 hours on 29 May after 38.5 hr of continuous leaf wetness. The average temperature during the interval was 11.7 C. Winds were light (0-1 m $s^{-1}$) until 0700 hours on 28 May, then increased slightly (0-3.5 m $s^{-1}$) until 1300 hours the same day. Winds were calm throughout the remainder of the infection period. Wind direction was initially from the E and NE, then shifted to SE and S at 1200 hours on 28 May.

**Infection period, 5-7 June (Plot E).** Light rain began at 1300 hours and continued until 1900 hours on 5 June. The leaves were dry by 1130 hours on 6 June, after 22.5 hr of continuous leaf wetness. The leaves were rewet by light showers at 1600 hours on 6 June, and remained wet until 0600 hours on 7 June. The length of the infection period including the 4.5 hr when the leaves were dry was 41 hr. The average temperature during the interval was 10.5 C. Winds were initially from

the SE until 2000 hours on 5 June, shifting to the NE and N for the remainder of the infection period. Wind speed throughout the period was light (0-2.7 m $s^{-1}$).

# Appendix E

## Plot design, the number of ascospores trapped and the number of lesions recorded for each plot in the Woodman Farm study, 1984 and 1985

This appendix contains the number of ascospores trapped and the number of lesions recorded for each primary infection period in a plot of trees left unprotected with fungicide for that infection period. The placement of volumetric spore traps in plots is presented in Figure 1. Figure 2 contains the number of ascospores trapped by each trap and the number of terminal and cluster lesions recorded for each tree for each infection period. Figures 2a, b, c, and d are data for the 1984 primary infection periods on 3-5 May, 8-9 May, 12-14 May, and 28 May - 2 June, respectively. Figures 2e, f, g, and h are data for the 1985 primary infection periods on 5-7 May, 18-19 May, 21-22 May, 27-29 May, and 5-7 June, respectively.

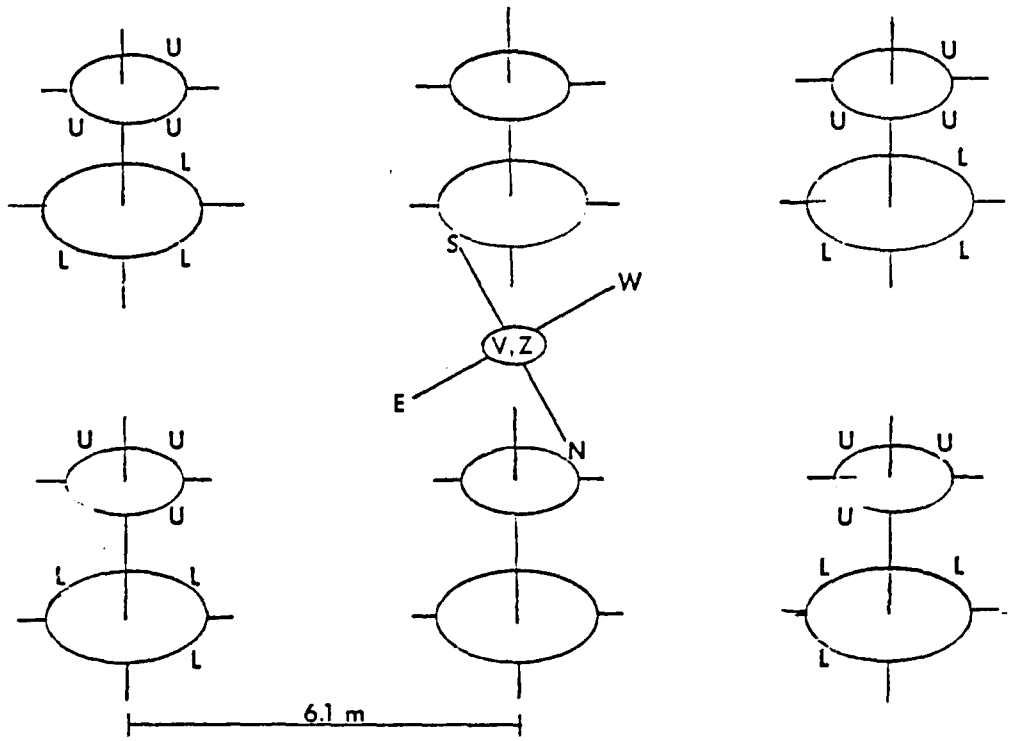



Figure 1. Location of spore traps within plots utilized in the 1984 and 1985 Woodman Farm study. Three "Zuck" volumetric spore traps were placed at 0.9 and 3.0 m in the lower (L) and upper (U) canopy of four trees, respectively. A 7-Day recording volumetric spore trap (V) and a "Zuck" volumetric spore trap (Z) were placed within an inoculum source (small elipse) containing approximately 1700 heavily scabbed leaves.

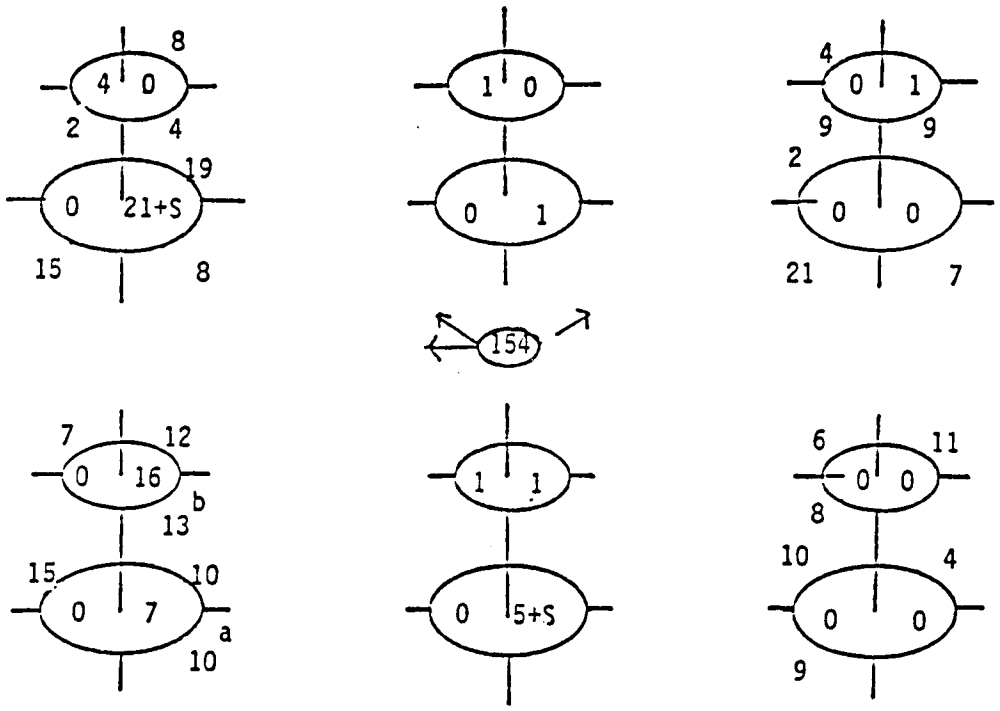



Figure 2a. Infection period of 3-5 May, 1984.

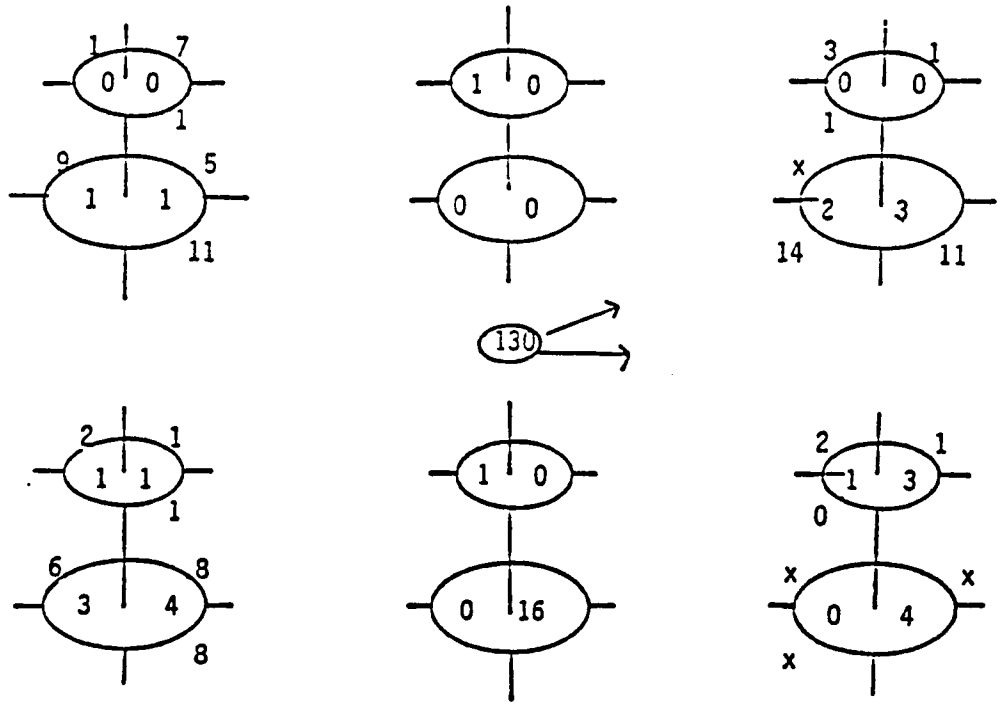



Figure 2b. Infection period of 8-9 May, 1984.

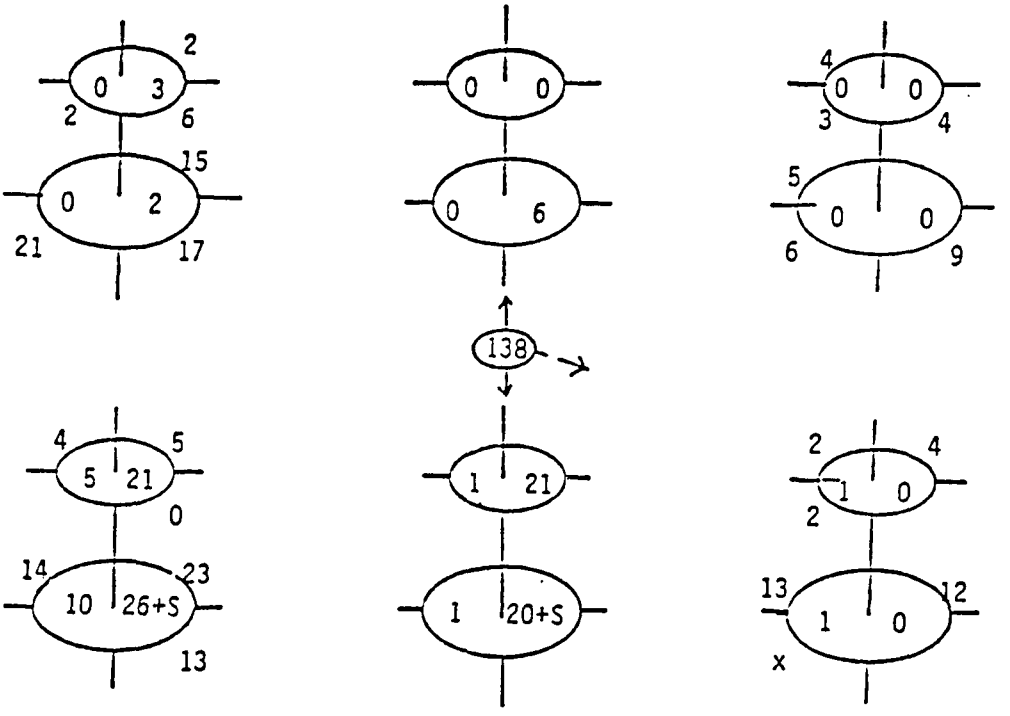



Figure 2c. Infection period of 12-14 May, 1984.

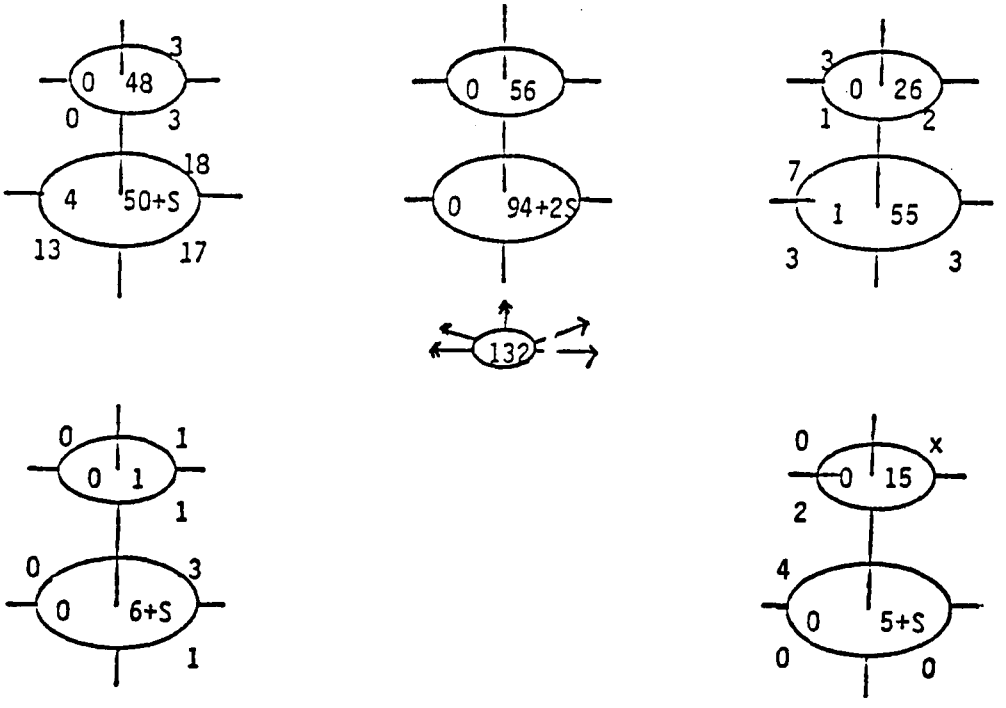



Figure 2d. Infection period of 28 May - 2 June, 1984.

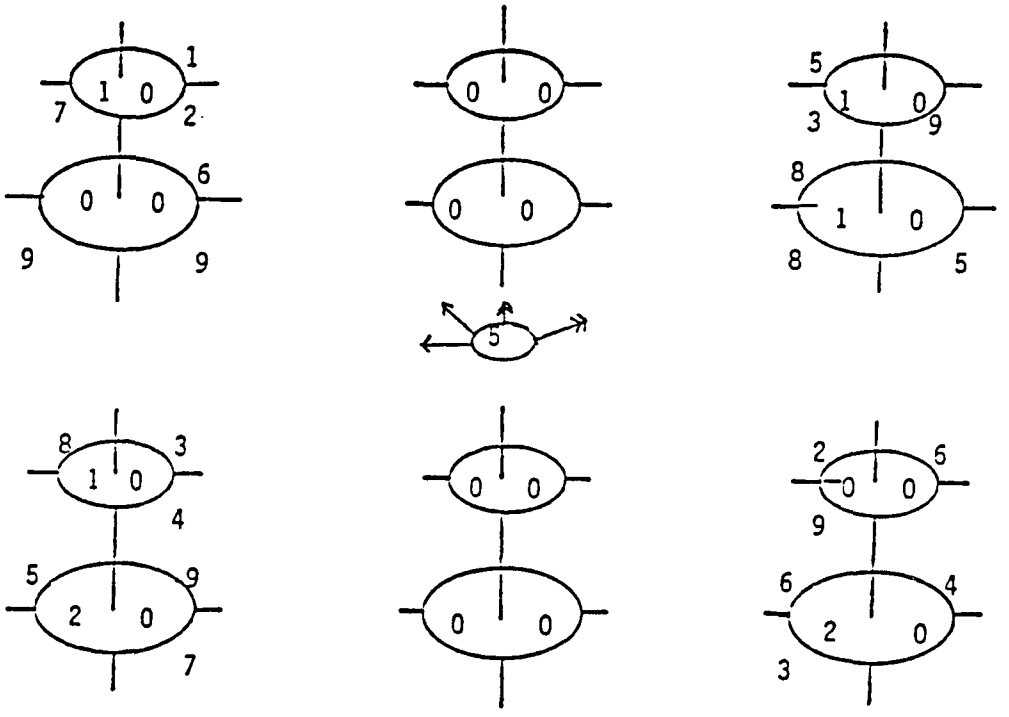


Figure 2e. Infection period of 5-7 May, 1985.

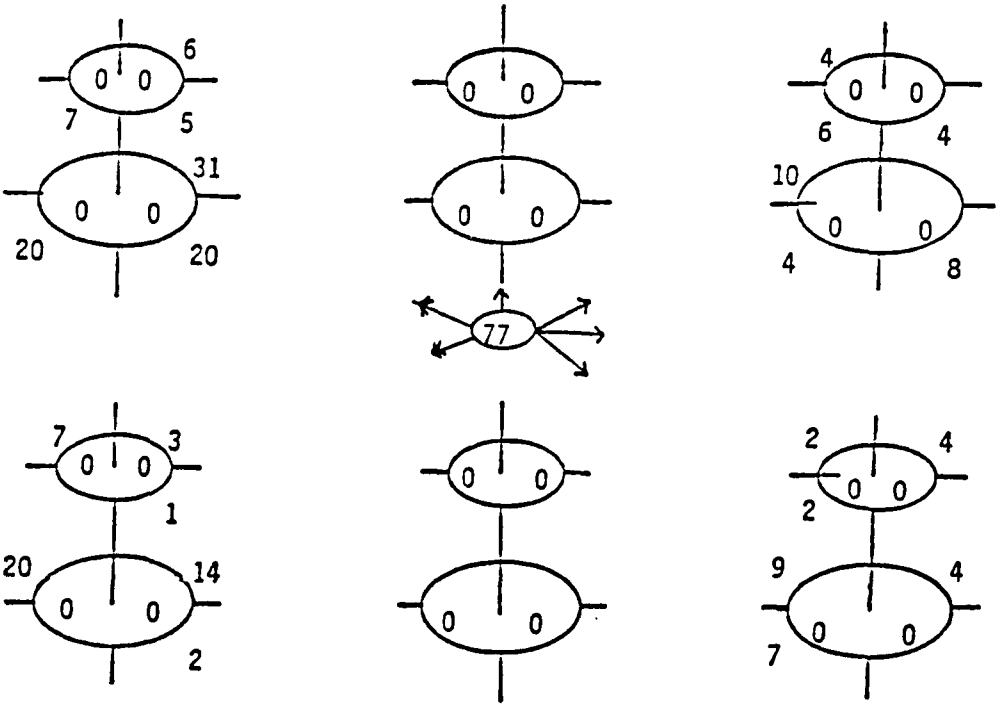


Figure 2f. Infection period of 18-19 May, 1985.

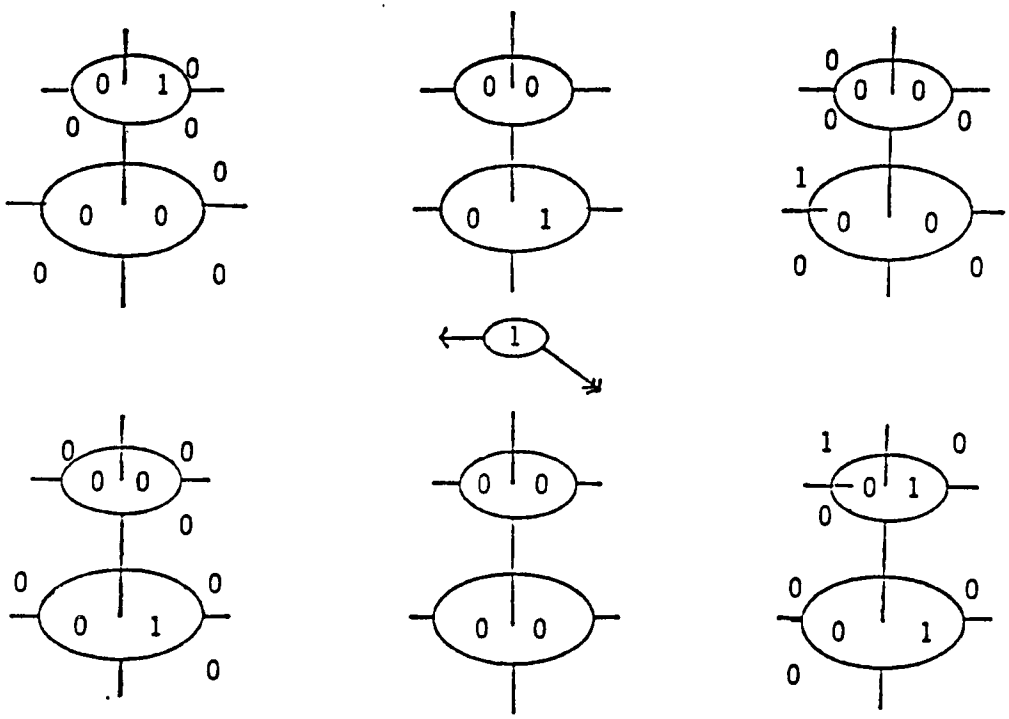


Figure 2g. Infection period of 21-22 May, 1985.

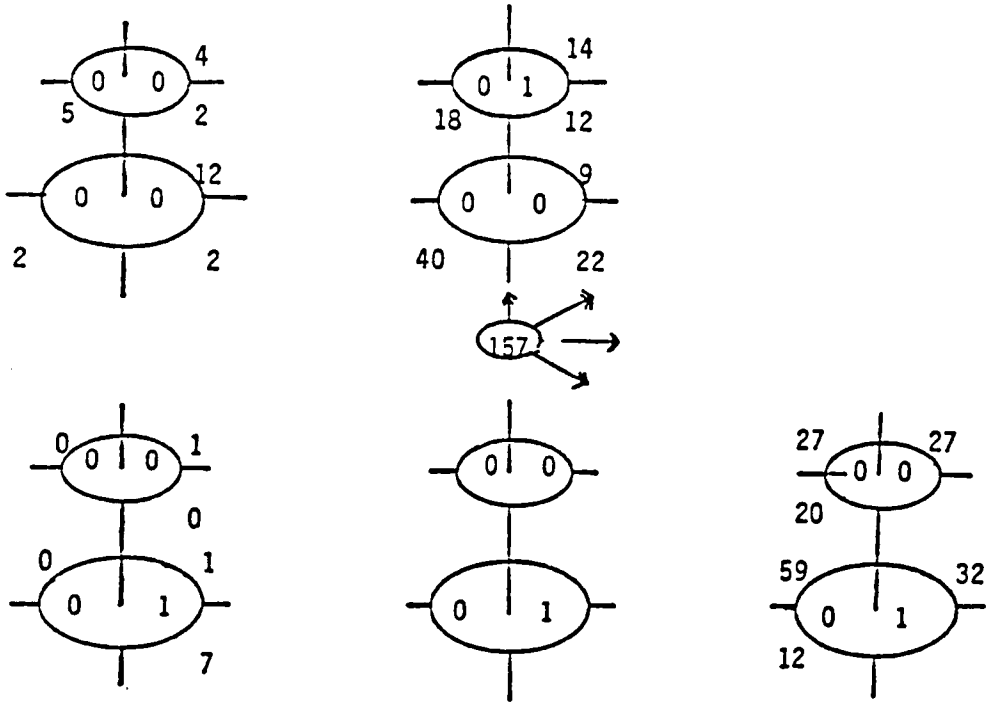


Figure 2h. Infection period of 27-29 May, 1985.

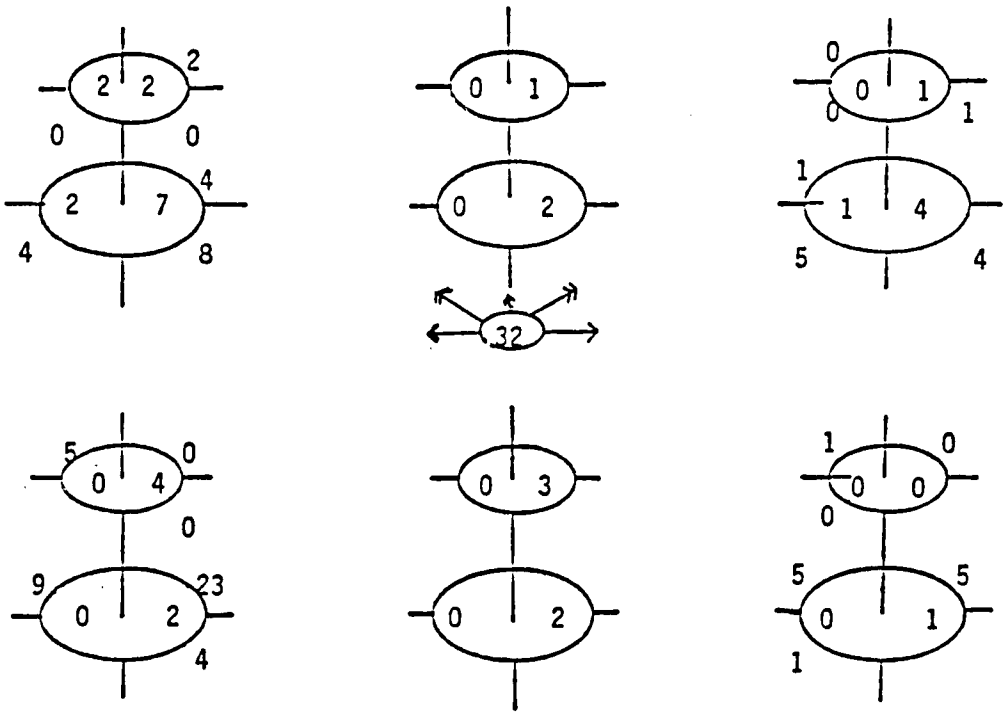



Figure 2i. Infection period of 5-7 June, 1985.

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Free at last
Free at last
God all mighty
Free at last!

Martin Luther King Jr.